FINAL BASELINE ECOLOGICAL RISK ASSESSMENT
PROBLEM FORMULATION

FOR THE
GULFCO MARINE MAINTENANCE
SUPERFUND SITE
FREEPORT, TEXAS

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JUNE 22, 2010

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LIST OF ACRONYMS

AET – apparent effects threshold

AST – aboveground storage tank

AUF – area-use factor (unitless)

BERA – Baseline Ecological Risk Assessment

COPEC – contaminants of potential ecological concern

CSM – conceptual site model

DDD – dichlorodiphenyldichloroethylene

DDE – dichlorodiphenyldichloroethane

DDT – dichlorodiphenyltrichloroethane

EPA – United States Environmental Protection Agency

ERL – effects range low

GRG – Gulfco Remediation Group

HPAH – high-molecular weight polynuclear aromatic hydrocarbon

HQ – hazard quotient

LPAH – low-molecular weight polynuclear aromatic hydrocarbon

NEDR – Nature and Extent Data Report

NOAEL – no-observed-adverse-effects-level

NPL – National Priorities List

PAH – polynuclear aromatic hydrocarbon

PCB – polychlorinated biphenyl

PSA – Potential Source Area

RI/FS – Remedial Investigation/Feasibility Study

SAP – Sampling and Analysis Plan

SLERA – Screening-Level Ecological Risk Assessment

SMDP – Scientific Management Decision Point

SOW – Statement of Work

TCEQ – Texas Commission on Environmental Quality

TSWQS – Texas Surface Water Quality Standard

UAO – Unilateral Administrative Order

USFWS – United States Fish and Wildlife Service

WP/SAP – Work Plan and Sampling and Analysis Plan

EXECUTIVE SUMMARY

The purpose of the Baseline Ecological Risk Assessment (BERA) problem formulation for the former Gulfco Marine Maintenance, Inc. site in Freeport, Brazoria County, Texas (the Site) is to use the Screening-Level Ecological Risk Assessment (SLERA) results and additional site-specific information to determine the scope and goals of the BERA.

Problem formulation includes the following:

- Refining the preliminary list of Contaminants of Potential Ecological Concern (COPECs) identified in the SLERA;
- Further characterizing the ecological effects of the refined COPEC list;
- Reviewing and refining information on contaminant fate and transport, complete exposure pathways, and ecosystems potentially at risk;
- Determining assessment endpoints (i.e., the specific ecological values to be protected); and
- Developing a conceptual site model with risk questions for the ecological investigation to address.

Steps were taken to refine the COPEC list (i.e., modification of conservative exposure assumptions and review of spatial COPEC distributions) and conduct literature research on the ecological effects of the refined list of COPECs, as well as their fate and transport characteristics relative to Site conditions. Subsequent to these steps, the following ecosystems have been identified as potentially at risk:

- Localized wetland areas in the North Area of the Site and north of the Site. The primary COPECs with hazard quotients (HQs) greater than one in wetland sediment are several polynuclear aromatic hydrocarbons (PAHs). Most of the PAH HQs exceedances are located in three areas: (1) a small area immediately northeast of the former surface impoundments; (2) a smaller area immediately south of the former surface impoundments; and (3) at a sample location in the southwest part of the North Area approximately 60 feet north of Marlin Avenue. Other COPECs include the organochlorine pesticides 4,4'-DDT, endrin aldehyde, and endrin ketone. Metals include arsenic, copper, lead, nickel, and zinc. Additionally, total acrolein and dissolved copper in wetland surface water in the first area (the area northeast of the former surface impoundments) exceed their respective ecological screening benchmark and Texas Surface Water Quality Standard (TSWQS). A small depression, identified as the pond, is

included in this exposure area and has 4,4'-DDT and zinc in the sediments and silver in the surface water.

- Localized areas of Intracoastal Waterway sediment within former Site barge slips. The predominant COPECs in these areas, as reflected by HQ exceedances, are also PAHs. The total PAH concentration was highest in the northernmost sample in the western barge slip. In the eastern barge slip, exceedances were limited to three PAHs, hexachlorobenzene (detected once), and the sum of high molecular weight PAHs (HPAHs) in one sample. 4,4'-DDT is the only organochlorine pesticide COPEC.

- Localized area of North Area soils south of the former surface impoundments. The organic COPECs in this area, where some buried debris was encountered in the shallow subsurface, are 4,4'-DDT and Aroclor-1254. Metals include barium, chromium, copper, and zinc.

The risk questions developed for these areas through the BERA Problem Formulation are:

Barge Slip and Wetland sediments: Does exposure to COPECs in sediment adversely affect the abundance, diversity, productivity, and function of sediment invertebrates?

Wetland surface water: Does exposure to COPECs in surface water adversely affect the abundance, diversity, productivity, and function of water-column invertebrates and fish?

North Area soils: Does exposure to COPECs in soil adversely affect the abundance, diversity, productivity, and function of soil invertebrates?

The approach for evaluating these risk questions, through the development and implementation of testable hypotheses and measures of effect and exposure based on this BERA problem formulation, will be described in the BERA Work Plan and Sampling and Analysis Plan (SAP).

1.0 INTRODUCTION

The United States Environmental Protection Agency (EPA) named the former site of Gulfco Marine Maintenance, Inc. in Freeport, Brazoria County, Texas (the Site) to the National Priorities List (NPL) in May 2003. The EPA issued a modified Unilateral Administrative Order (UAO), effective July 29, 2005, which was subsequently amended effective January 31, 2008. The UAO required Respondents to conduct a Remedial Investigation and Feasibility Study (RI/FS) for the Site. Pursuant to Paragraph 37(d)(x) of the Statement of Work (SOW) for the RI/FS, included as an Attachment to the UAO, a Final Screening Level Ecological Risk Assessment (SLERA) was prepared by Pastor, Behling & Wheeler, LLC (PBW), on behalf of LDL Coastal Limited LP (LDL), Chromalloy American Corporation (Chromalloy) and The Dow Chemical Company (Dow), collectively known as the Gulfco Restoration Group (GRG) (PBW, 2010a). The Scientific/Management Decision Point (SMDP) provided in the Final SLERA concluded that the information presented therein indicated a potential for adverse ecological effects, and a more thorough assessment was warranted. A Draft Baseline Ecological Risk Assessment (BERA) Problem Formulation was prepared by PBW, consistent with Paragraphs 37(d)(xi) and (xii) of the UAO as the next step in that assessment (PBW, 2010b). This Final BERA Problem Formulation report has been prepared by URS Corporation (URS) based on comments received from the EPA and Texas Commission on Environmental Quality (TCEQ).

Figure 1 provides a map of the Site vicinity, while Figure 2 provides a Site map.

1.1 REPORT PURPOSE

The ecological risk assessment process is outlined in the SOW (Page 20, Paragraphs 37(d)(xi) and (xii)). A diagram of the process as provided in EPA's Ecological Risk Assessment Process for Superfund (EPA, 1997) is provided in Figure 3. Problem formulation represents the third step in the eight-step ecological risk assessment process. The purpose of the problem-formulation phase is to refine the screening level problem formulation, and use the SLERA results and additional site-specific information to determine the scope and goals of the BERA.

As described in EPA, 1997, problem formulation includes the following:

- Refining the preliminary list of COPECs identified in the SLERA;
- Further characterizing the ecological effects of the refined COPEC list;

- Reviewing and refining information on contaminant fate and transport, complete exposure pathways, and ecosystems potentially at risk;
- Determining specific assessment endpoints (i.e., the specific ecological values to be protected); and
- Developing a conceptual model with risk questions that the ecological investigation will address.

The SMDP at the end of problem formulation is the identification and agreement on the conceptual model, including assessment endpoints, exposure pathways, and questions or risk hypotheses. The results of this SMDP are then used to select measurement endpoints for development of the BERA Work Plan and Sampling & Analysis Plan (Work Plan/SAP).

1.2 SITE BACKGROUND

1.2.1 Site Description

The Site is located in Freeport, Texas at 906 Marlin Avenue (also referred to as County Road 756) (Figure 1). The Site consists of approximately 40 acres along the north bank of the Intracoastal Waterway between Oyster Creek (approximately one mile to the east) and the Texas Highway 332 bridge (approximately one mile to the west). The Site includes approximately 1,200 feet (ft.) of shoreline on the Intracoastal Waterway, the third busiest shipping canal in the US (TxDOT, 2001) that, on the Texas Gulf Coast, extends 423 miles from Port Isabel to West Orange.

Marlin Avenue divides the Site into two primary areas (Figure 2). For the purposes of descriptions in this report, Marlin Avenue is approximated to run due west to east. The property to the north of Marlin Avenue (the North Area) consists of undeveloped land and closed surface impoundments, while the property south of Marlin Avenue (the South Area) was developed for industrial uses with multiple structures, a dry dock, sand blasting areas, an aboveground storage tank (AST) tank farm, and two barge slips connected to the Intracoastal Waterway. The South Area is zoned as "W-3, Waterfront Heavy" by the City of Freeport. This designation provides for commercial and industrial land use, primarily port, harbor, or marine-related activities. The North Area is zoned as "M-2, Heavy Manufacturing."

Adjacent property to the north, west, and east of the North Area is undeveloped. Adjacent property to the east of the South Area is currently used for industrial purposes while to the west

the property is currently vacant and previously served as a commercial marina. The Intracoastal Waterway bounds the Site to the south. Residential areas are located south of Marlin Avenue, approximately 300 feet west of the Site, and 1,000 feet east of the Site.

The Intracoastal Waterway is a major corridor for commercial barge traffic and other boating activities. Approximately 50,000 commercial vessel trips and 28 million short tons of cargo were transported on the Galveston to Corpus Christi section of the Intracoastal Waterway in 2006. The vast majority of this cargo (greater than 23 million tons) was petroleum, chemicals or related products (USACE, 2006). The Intracoastal Waterway design width and depth in the vicinity of the Site, based on USACE mean low tide datum, is 125 feet wide and 12 feet deep (USACE, 2008). The waterway is maintained by periodic dredging operations conducted by the USACE as frequently as every 20 to 38 months, and as infrequently as every 5 to 46 years (Teeter et al., 2002). A September 2008 survey indicated that actual channel depths in the 19-mile reach from Chocolate Bayou to Freeport Harbor, which includes the Site vicinity, ranged from 9.3 to 11.1 feet (USACE, 2008). According to the USACE (USACE, 2009), the Intracoastal Waterway in the immediate vicinity of the Site is not currently scheduled for dredging, although dredging is performed approximately every three to four years and the area to the west near Freeport Harbor (Intracoastal Waterway Mile 395) was dredged in 2009.

The South Area includes approximately 20 acres of upland that was created from dredged material from the Intracoastal Waterway. The two most significant surface features within the South Area are a Former Dry Dock and the AST Tank Farm (Figure 2). The remainder of the South Area surface consists primarily of former concrete laydown areas, concrete slabs from former Site buildings, gravel roadways and sparsely vegetated open areas with some localized areas of denser brush vegetation, particularly near the southeast corner of the South Area.

Some of the North Area is upland created from dredge spoil, but most of this area is considered wetlands, as per the United States Fish and Wildlife Service (USFWS) Wetlands Inventory Map (Figure 4) (USFWS, 2008). This wetland area generally extends from East Union Bayou to the southwest, to the Freeport Levee to the north, to Oyster Creek to the east (see Figure 1). The most significant surface features in the North Area are two ponds (the Fresh Water Pond and the Small Pond) and the closed former surface impoundments. The former surface impoundments and the former parking area south of the impoundments and Marlin Avenue comprise the vast majority of the upland area within the North Area (Figure 4).

Field observations during the RI indicate that the North Area wetlands are irregularly flooded with nearly all of the wetland area inundated by surface water that can accumulate to a depth of one foot or more during extreme high tide conditions, storm surge events, and/or in conjunction with surface flooding of Oyster Creek northeast of the Site (Figure 1). Due to a very low topographic slope and low permeability surface sediments, the wetlands are also very poorly draining and can retain surface water for prolonged periods after major rainfall events. Under normal tide conditions and during periods of normal or below normal rainfall, standing water within the wetlands (outside of the two ponds discussed below) is typically limited to a small, irregularly shaped area immediately north of the Fresh Water Pond and a similar area immediately south of the former surface impoundments (see Figure 2). Both of these areas can be completely dry, as was observed in June 2008. As such, given the absence of any appreciable areas of perennial standing water, the wetlands are effectively hydrologically isolated from Oyster Creek, except during intermittent, and typically brief, flooding events.

The Fresh Water Pond is approximately 4 to 4.5 feet deep and is relatively brackish (specific conductance of approximately 40,000 umhos/cm and salinity of approximately 25 parts per thousand). This pond appears to be a borrow pit created by the excavation of soil and sediment as suggested by the well-defined pond boundaries and relatively stable water levels. Water levels in the Fresh Water Pond are not influenced by periodic extreme tidal fluctuations as the pond dikes preclude tidal floodwaters in the wetlands from entering the pond, except for extreme storm surge events, such as observed during Hurricane Ike in September 2008.

The Small Pond is a very shallow depression located in the eastern corner of the North Area. The Small Pond is not influenced by daily tidal fluctuations and behaves in a manner consistent with the surrounding wetland, i.e., becomes dry during dry weather, but retains water in response to and following rainfall and extreme tidal events. Relative to the Fresh Water Pond, water in the Small Pond is less brackish based on specific conductance (approximately 14,000 umhos/cm) and salinity (approximately eight parts per thousand) measurements.

1.2.2 Site History

A detailed discussion of Site operational history was provided in the RI/FS Work Plan (PBW, 2006). Key elements of that discussion are noted herein. During the 1960s, the Site was used for

occasional welding but there were no on-site structures (Losack, 2005). According to the Hazard Ranking Score Documentation (TNRCC, 2002), from 1971 through 1999, at least three different owners used the Site as a barge cleaning facility. Beginning in approximately 1971, barges were brought to the facility and cleaned of waste oils, caustics and organic chemicals, with these products stored in on-site tanks and later sold (TNRCC, 2002). Sandblasting and other barge repair/refurbishing activities also occurred on the Site. At times during the operation, wash waters were stored either on a floating barge, in on-site storage tanks, and/or in surface impoundments on Lot 56 of the Site. The surface impoundments were closed under the Texas Water Commission's (Texas Commission on Environmental Quality (TCEQ) predecessor agency) direction in 1982 (Carden, 1982).

Aerial spraying of the wetland areas north of Marlin Avenue, including the North Area, for mosquito control has historically been and continues to be performed by the Brazoria County Mosquito Control District and its predecessor agency, the Brazoria County Mosquito Control Department (both referred to hereafter as BCMCD). Aerial spraying for mosquito control has been performed over rural areas in the county since 1957 (Lake Jackson News, 1957). Historically, aerial spraying of a DDT solution in a "clinging light oil base" was performed from altitudes of 50 to 100 feet (Lake Jackson News, 1957). Recently BCMCD has been using Dibrom®, an organophosphate insecticide, with a diesel fuel carrier through a fogging atomizer application (Facts, 2006, 2008a, 2008b). Truck-based spraying has also been performed along Marlin Avenue. Both types of spraying were observed during the performance of Site RI activities.

1.3 REPORT ORGANIZATION

The organization for this report has been patterned after that suggested in EPA guidance (EPA, 1997). As such, Section 2.0 provides a refinement of the COPECs indentified in the SLERA. Section 3.0 characterizes the potential ecological effects of that refined list of COPECs. Section 4.0 describes significant fate and transport characteristics, ecosystems potentially at risk and complete exposure pathways. Section 5.0 describes assessment endpoints, and Section 6.0 provides the refined Conceptual Site Model and resulting risk decisions. The problem formulation SMDP is discussed in Section 7.0. Appendix A contains a table from the SLERA listing COPECs and media recommended for further evaluation in the BERA. Appendix B

presents environmental fate/transport and toxicological profiles for the COPECs identified in Table 29 of the Final 2010 SLERA (PBW, 2010a).

2.0 REFINEMENT OF CONTAMINANTS OF POTENTIAL ECOLOGICAL CONCERN

The Final SLERA (PBW, 2010a) concluded with the SMDP that there is a potential for adverse ecological effects from COPECs and a more thorough assessment through continuation of the ecological risk assessment process was warranted. The Final SLERA calculated HQs based on conservative screening-level assumptions, such as area-use factors (AUFs) of 100%, 100% contaminant bioavailability, maximum ingestion rates, and minimum body weights. Appendix A provides the SLERA table identifying COPECs with HQs greater than one.

As illustrated in Appendix A (Table 29 from the Final SLERA), the screening-level evaluation identified HQs greater than one for the following Site media and receptors:

- Invertebrate receptors in South Area soils (as represented by the earthworm);
- Invertebrate receptors in North Area soils (also represented by the earthworm);
- Benthic receptors in Site Intracoastal Waterway sediment (as represented by the polychaetes *Capitella capitata*);
- Benthic receptors in Site wetlands sediment (as represented by the polychaetes *Capitella capitata*);
- Invertebrate receptors in wetlands surface water (as represented by the fiddler crab *Uca rapax* and killifish *Fundulus grandis*);
- Benthic receptors in Site pond sediment (as represented by the polychaetes *Capitella capitata*); and
- Invertebrate receptors in pond surface water (as represented by the fiddler crab *Uca rapax* and killifish *Fundulus grandis*).

The Final SLERA (PBW, 2010a) concluded that upper trophic level receptors were not at risk from these COPECs.

2.1 REFINEMENT PROCEDURES

As described in EPA, 1997, the purpose of the refinement step of problem formulation is to consider how the HQs in the SLERA would change when more realistic conservative

assumptions are used. As previously discussed, the Final SLERA (PBW, 2010a) concluded that upper trophic level (non-sedentary) receptors are not at risk from COPECs.

2.2 ASSESSMENT OF EXPOSURE POTENTIAL OF SOUTH AREA SOILS

The South Area of the Site is characterized by the following habitat-related considerations:

1. It is zoned by the City of Freeport as "W-3, Waterfront Heavy", which provides for commercial and industrial land use, primarily port, harbor, or marine-related activities;
2. A restrictive covenant placed on the deed ensures that future land use for this parcel of land is commercial/industrial;
3. The area does not serve as valuable habitat, foraging area, or refuge for ecological communities, including threatened/endangered or otherwise protected species;
4. The area does not contain consistent and contiguous habitat but, rather, the area is broken up by the presence of concrete slabs, pads, and driveways;
5. The area only exhibits minimal natural functions because of the disturbed nature of the land due to the industrial use of the property and adjacent properties; and
6. There are minimal if any attractive features at the South Area that would support a resident wildlife community.

Since the Site was developed in the early 1960s, as described in the Nature and Extent Data Report (PBW, 2009), it has been used for industrial purposes. It is also bounded by former and/or current industrial properties to the east and west. The Site has not been used since approximately 1999 and opportunistic grasses and small shrubs have grown on some portions of the Site that do not have concrete, oyster shell, or gravel cover. The Site will most certainly be used in the future for industrial purposes since the barge slips are valuable to many types of businesses in the area, and it is very unlikely that any portion of the Site will return to "natural" conditions.

The earthworm was chosen as the receptor of concern in the Final SLERA (PBW, 2010) for the detritivores and soil invertebrates at the Site. The only HQs greater than one for South Area soils in the SLERA, using maximum soil concentrations for the soil invertebrate receptor, were for the following compounds: 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, Aroclor-1254, barium, chromium, copper, zinc, and total high molecular weight PAHs (Total HPAHs). While the SLERA used maximum concentrations to calculate HQs for sedentary receptors, using the 95th percentile upper

confidence limit on the mean (95% UCL) for these compounds results in HQs less than one for all compounds except barium and zinc. All HQs for higher trophic level organisms were less than one for South Area soils. These HQs were calculated using 95% UCLs as the exposure point concentration.

Figure 2 shows the areas of concrete slabs, pads, and driveways in the South Area of the Site. Although these areas are not physically contiguous, these areas combined account for roughly 28% of the surface area of the South Area. For the remaining 72% of the South Area, soil borings were advanced during the RI at 85 locations. Shallow soils at 73 of the 85 boring locations (86%) were characterized as containing either compacted fill material (typically described as varying combinations of sand, clay, gravel, oyster shell, and/or brick fragments) or firm clays (that would be difficult for earthworms to burrow in) within the upper two feet of the subsurface. Based on the amount of surface area not covered by concrete slabs, pads, and driveways (72%) and the fraction of the area not comprised of fill material or firm clays (14% as indicated by the soil descriptions obtained from the South Area boring logs), an estimated 10% of the South Area (14% of 72%) has soils that could be considered potential ecological habitat for earthworms. This area equates to roughly 2 acres. These areas of potential soil invertebrate habitat are not contiguous. Additionally, the zinc HQs at three of the soil locations considered potential ecological habitat for earthworms were below one.

The evidence discussed in the paragraphs above indicate that the South Area soils do not represent a valuable ecological resource that warrants further evaluation in order to protect invertebrates such as earthworms and, therefore, there is no further assessment of the South Area soils.

2.3 SPATIAL DISTRIBUTION OF COPECs IN REMAINING AREAS

In order to evaluate potential hotspots and the spatial distributions of the remaining COPECs, HQ exceedances in individual samples are plotted by environmental medium in Figures 5 through 9. For soils, the HQs are based on no-observed-adverse-effects-levels (NOAELs). For sediments, HQs are based on marine benchmarks (e.g., Effects Range-Low [ERL]) from TCEQ (2006) where available, or other sediment quality guidelines (e.g., Apparent Effects Thresholds [AET]) from Buchman (2008). The paragraphs below discuss the spatial trends of the HQ exceedances

observed in the figures for the North Area soils, Intracoastal Waterway Sediment, Wetlands Sediment, and Pond Sediment. The listing of COPECs is presented in Appendix A.

Figure 5 shows HQ exceedances for soil invertebrates in the North Area. For the organic COPECs, the only HQ exceedances are 4,4'-DDT and Aroclor-1254 in the 1.5 to 2.0 foot depth interval sample from SB-204. This boring was located in an area where buried debris was observed and some of this debris (painted wood fragments and rubber) was observed in this specific sample interval. Barium, chromium, copper, and zinc were detected above screening levels in several locations. The maximum detection of zinc is from SB-202 located in the same debris area as SB-204.

Figure 6 shows HQ exceedances for benthic receptors in Site Intracoastal Waterway sediment. None of the HQs are greater than 5 and 75 percent are less than or equal to 2. As indicated on this figure, the HQs greater than one are nearly all PAHs, except for 4,4'-DDT in a sample next to the western boundary of the Site and hexachlorobenzene on the edge of the eastern barge slip, and most are associated with samples in the northern end of the western barge slip.

Figure 7 shows HQ exceedances for benthic receptors in Site wetland sediment. As shown in this figure, the predominant and highest HQs are associated with PAHs (both individual PAHs and low molecular weight PAHs (LPAH), HPAH, and total PAHs). Most of the PAH HQ exceedances are located in three areas: (1) a small area immediately northeast of the former surface impoundment (where most of the highest PAH HQs are observed; e.g., 2WSED2); (2) a smaller area immediately south of the former surface impoundments (e.g., 2WSED17); and (3) at sample location NB4SE08 in the southwest part of the North Area. The three highest HQs, all located in the area north of the former surface impoundments, are for dibenz(a,h)anthracene. Arsenic, copper, lead, nickel, and zinc were all detected at concentrations greater than their ERLs.

Figure 8 shows HQ exceedances for benthic receptors in pond sediment. As shown in this figure, the sole organic HQ exceedance is for 4,4'-DDT in the southernmost sample from the Small Pond. Zinc was detected in the sediments above the ERL in three locations.

There are three COPECs, total acrolein, dissolved copper, and dissolved silver, with maximum concentrations that exceed their respective ecological screening benchmark and TSWQS. Acrolein was detected once in four surface water samples from the wetlands area, and not

detected in any other Site samples. Dissolved copper was detected in three of four surface water samples from the wetlands area. All of the detections are greater than the TSWQS, the highest being about three times greater. Both acrolein and dissolved copper are retained for further evaluation in the BERA. Silver was detected in the pond surface water above the TSWQS and is retained as a COPEC.

3.0 CHARACTERIZATION OF ECOLOGICAL EFFECTS

The Final SLERA (PBW, 2010a) included a literature search of potential ecological effects from the initial COPECs. As part of problem formulation in the BERA, additional literature information related to the remaining Site COPECs was obtained and reviewed.

The Final SLERA (PBW, 2010a) concluded that upper trophic level receptors were not at risk from these COPECs. For sediment and soil invertebrates, benchmarks (e.g., ERLs for sediment) from TCEQ (2006) were used. If a marine/estuarine benchmark was not available, sediment quality guidelines from Buchman (2008) were selected. For soil, the TCEQ (2006) benchmarks for the protection of earthworms were used.

A number of researchers have performed studies to determine AETs, which are measures of sediment effect levels developed using the empirical data from the results of toxicity tests and benthic community structure. They are derived by determining, for a given chemical within a data set, the chemical sediment concentration above which a particular adverse biological effect is always statistically significant relative to a designated reference location.

ERLs and ERMs are also statistically-derived sediment benchmark values based on a variety of benthic endpoints including mortality, community structure, reproductive, and other effects. These sediment quality guidelines are intended as informal (i.e., non-regulatory) benchmarks to aid in the interpretation of chemical data. Low-range values (i.e., ERLs) are intended as concentrations below which adverse effects upon sediment-dwelling fauna would be expected only infrequently. ERMs, on the other hand, are intended to represent chemical concentrations above which adverse effects are likely to occur (Long and MacDonald, 1998).

4.0 CONTAMINANT FATE AND TRANSPORT AND ECOSYSTEMS POTENTIALLY AT RISK

The Final SLERA (PBW, 2010a) included a preliminary evaluation of contaminant fate and transport, ecosystems potentially at risk, and complete exposure pathways for COPECs and media that might pose an adverse risk to terrestrial and aquatic receptors. The exposure pathways and ecosystems associated with the assessment endpoints carried forward from the SLERA were evaluated in more detail in this problem formulation. Consistent with EPA (1997), this evaluation also considered the possible reduction of potentially complete, but less significant, exposure pathways to examine the critical exposure pathways, where appropriate. The findings of this evaluation are presented below.

4.1 CONTAMINANT FATE AND TRANSPORT

Additional information was acquired from the scientific literature regarding the fate and transport of the remaining COPECs. Specifically, details about transport mechanisms in terrestrial and aquatic systems similar to those found at the Site were obtained and are discussed below.

4.1.1 Potential Transport Mechanisms in Terrestrial Systems

Potentially significant routes of migration for Site COPECs relative to terrestrial systems occur in the primary transport media of air and surface water (runoff). Surface water runoff, or overland flow, can carry dissolved COPECs in solution or move COPECs adsorbed to soil particles from one portion of the Site to another, depending on surface topography. The same mechanisms described for overland flow in the wetlands (Section 4.1.2) apply to the upland areas of the North Area. Airborne transport of Site COPECs is possible via entrainment of COPEC-containing particles in wind. This pathway is a function of particle size, chemical concentrations, moisture content, degree of vegetative cover, surface roughness, size and topography of the source area, and meteorological conditions (wind velocity, wind direction, wind duration, precipitation, and temperature). Movement of airborne contaminants occurs when wind speeds are high enough to dislodge particles; higher wind velocities are required to dislodge particles than are necessary to maintain suspension.

4.1.2 Potential Transport Mechanisms in Estuarine Wetland and Aquatic Systems

Potentially significant routes of migration for Site COPECs relative to wetland and aquatic systems occur in the primary transport media of surface water and sediment. The primary surface water/sediment pathways for potential contaminant migration from Site potential source areas (PSAs) are: (1) erosion/overland flow to wetland areas north and east of the Site from the North Area due to rainfall runoff and storm/tide surge; and (2) erosion/overland flow to the Intracoastal Waterway from the South Area as a result of rainfall runoff and extreme storm surge/tidal flooding events.

The primary North Area PSAs, the former surface impoundments, were closed and capped in 1982. Thus, potential migration from these areas to the adjacent wetlands would have to have occurred during the operational period of the impoundments, potentially when discharges from the impoundments in July 1974 and August 1979 reportedly "contaminated surface water outside of ponds" and "damaged some flora north of the ponds" (EPA, 1980). Although not associated with Site operations, the historical and ongoing spraying of pesticides in the wetland areas for mosquito control could represent a potential source of DDT and PAHs (associated with the light oil base and diesel carrier used in spraying then and now, respectively) to the wetlands.

Overland flow during runoff events occurs in the direction of topographic slope. Overland flow during runoff events occurs if soils are fully saturated and/or precipitation rates are greater than infiltration rates; therefore, this type of flow is usually associated with significant rainfall events. As a result of the minimal slope at the site, overland flow during more routine rainfall events is generally low, with runoff typically ponding in many areas of the Site. Extreme storm events, such as Hurricane Ike in September 2008, can inundate the Site, resulting in overland flow during both storm surge onset and recession. During less extreme storm surge events or unusually high tides, tidal flow to wetland areas on and adjacent to the Site occurs from Oyster Creek northeast of the Site (Figure 1); however, the wetland areas are more typically hydrologically isolated from Oyster Creek.

Potential contaminant migration in surface water runoff can occur as both sediment load and dissolved load; therefore, both the physical and chemical characteristics of the contaminants are important with respect to surface-water/sediment transport. The low topographic slope of the Site and adjacent areas is not conducive to high runoff velocities or high sediment loads.

Consequently, surface soil particles would not be readily transported in the solid phase. Additionally, the vegetative cover in the North Area is not conducive to significant soil erosion and resulting sediment load transport with surface water in these areas. Dissolved loads associated with surface runoff from the North Area would likewise be expected to be minimal due to the aforementioned absence of exposed PSAs, and the relatively low solubilities of those COPECs (primarily, pesticides and PAHs) that are present.

4.1.3 COPEC-Specific Fate and Transport Characteristics

PAHs. A detailed literature review related to PAH fate and transport characteristics in similar settings to the Site was performed for the ecological problem formulation for the Alcoa (Point Comfort)/Lavaca Bay Superfund Site (Alcoa, 2000). That document (used with permission) provided significant parts of the summary presented herein. Due to their low solubility and relatively high affinity for adsorption to soils, sediment organic matter, PAHs in the aquatic environment are primarily associated with particulate matter and sediments (Neff, 1985). PAHs sorb to both inorganic and organic surfaces, although adsorption to organic surfaces tends to be most important. PAH adsorption to particulate mater, especially HPAHs, is a primary mechanism for removing these compounds from the water column, resulting in subsequent deposition to sediments. PAH sorption to sediments is strongly influenced by sediment organic carbon content. PAH sorption is also influenced by particle size (Karickhoff et al., 1979); the smaller the particle size, the greater the adsorption potential.

Benthic organisms accumulate PAHs by two primary exposure routes: (1) bioconcentration through transport across biological membranes exposed to aqueous phase PAHs (i.e., pore water); and (2) bioaccumulation through direct food or sediment ingestion. For benthic organisms, direct ingestion of food and/or sediments is often the most significant exposure pathway for HPAHs (Niimi and Dookhran, 1989; Eadie et al., 1985; Weston, 1990), while pore water is likely a more significant route for LPAH accumulation (Meador et al., 1995b; Adams, 1987; Landrum, 1989). Differences in feeding regime (i.e., epibenthic, infaunal) also influence which exposure route is most significant.

As a result of these issues, PAH accumulation by benthic organisms can vary. In addition, the degree to which organisms accumulate PAHs depends on their ability to metabolize these compounds. Although some organisms metabolize PAHs (e.g., fish and mammals), many benthic

invertebrates are limited in their ability to metabolize PAHs (Meador et al., 1995a; Landrum, 1982; Frank et al., 1986).

In general, there is little evidence to suggest PAHs biomagnify in aquatic systems. However, because of the limited ability of invertebrates to metabolize PAHs, some biomagnification may occur in lower trophic levels (Meador et al., 1995a; McElroy et al., 1989; Broman et al., 1990; Suede et al., 1994). Although metabolism often results in detoxification, some PAH metabolites are more toxic than parent materials; however, the degree to which these metabolites are accumulated by aquatic organisms is unknown.

Organochlorine Pesticides and PCBs. Organochlorine pesticides and PCBs are of interest in characterizations of risk to ecological receptors due to the affinity of these compounds to sorb tightly onto soils and sediments and persist for long periods of time in the environment. The degradation of organochlorine compounds in the environment is dependent on the degree and pattern of chlorination, with compounds possessing five or more chlorine atoms more persistent in the environment than those with fewer chlorine atoms.

Benthic invertebrate communities are particularly susceptible to organochlorine compound impacts as consequence of ingestion of sediment particles and exchange of PCBs directly from the particles. The silt and clay content of sediments can have a significant influence on the bioavailability of organochlorine compounds, with low silt and clay content sediments exhibiting decreased effects on benthic communities (Eisler, 1986). Due to bioaccumulative properties, organochlorine compounds cycle readily from sediment sources into upper trophic levels. This class of compounds are soluble in lipids and partition readily into the fatty tissues of higher-level consumers, with the ability to be metabolized decreasing as the number of substituted chlorines increases. For highly substituted compounds, metabolism is less likely and accumulation may continue indefinitely. The fate of organochlorine compounds within biologic systems is wide ranging as a result of differences in the ability to accumulate, metabolize, and eliminate specific isomers.

4.2 ECOSYSTEMS POTENTIALLY AT RISK

Based on the COPECs and media recommended for further evaluation in Table 1, and in consideration of the ecological effects literature evaluation (Section 3.0), the fate and transport characteristics (Section 4.1), and the nature of the ecosystems themselves, the following ecosystems have been identified as potentially at risk:

- Localized wetland areas in the North Area and north of the Site. The primary COPECs with HQ exceedances in wetland sediment are several PAHs (Table 2). As shown on Figure 7, most of the PAH HQs are located in three areas: (1) a small area immediately northeast of the former surface impoundments (where most of the highest PAH HQs are observed; e.g., 2WSED2); (2) a smaller area immediately south of the former surface impoundments (e.g., 2WSED17); and (3) at sample location NB4SE08 in the southwest part of the North Area approximately 60 feet north of Marlin Avenue. Other COPECs include the organochlorine pesticides 4,4'-DDT, endrin aldehyde, and endrin ketone. Metals include arsenic, copper, lead, nickel, and zinc. Additionally, total acrolein and dissolved copper in wetland surface water in the first area (the area northeast of the former surface impoundments) exceed their respective surface water benchmark and TSWQS. A small depression, identified as the pond, is included in this exposure area and has 4,4'-DDT and zinc in the sediments and silver in the surface water.

- Localized areas of Intracoastal Waterway sediment within the former barge slips. The predominant COPECs in these areas, as reflected by HQ exceedances, are PAHs. The total PAH concentration (5.62 mg/kg) was highest in the northernmost sample in the western barge slip. In the eastern barge slip, exceedances were limited to three PAHs , hexachlorobenzene (detected once), and HPAHs in one sample. 4,4'-DDT is the only organochlorine pesticide COPEC.

- Localized area of North Area soils south of the former surface impoundments. As previously described (Section 2.3), for organic COPECs, the only HQ exceedances are 4,4'-DDT and Aroclor-1254 in the 1.5 to 2.0 foot depth interval sample from SB-204. This boring was located in an area where buried debris was observed and some of this debris (painted wood fragments and rubber) was observed in this specific sample interval. Metals include barium, chromium, copper, and zinc.

5.0 SITE-SPECIFIC ASSESSMENT ENDPOINTS

Assessment endpoints are explicit expressions of the ecological resource to be protected for a given receptor of potential concern (EPA, 1997). Several assessment endpoints were identified in the SLERA to focus the screening evaluation on relevant receptors rather than attempting to evaluate risks to all potentially affected ecological receptors. As part of this BERA problem formulation, these assessment endpoints were re-evaluated based on the remaining environmental media and receptors of potential concern.

5.1 TERRESTRIAL ASSESSMENT ENDPOINTS

The terrestrial portion associated with the Site that remains of concern is a small area of land south of the former surface impoundments. The environmental value of upland lands is related to its ability to support plant communities, soil microbes/detritivores, and wildlife. Based on the steps taken in the refinement (Section 2.0) and new information obtained about COPEC fate and transport and ecosystems at risk (Section 4.0), the following remains the assessment endpoint for the BERA (Table 2):

- *Soil invertebrates abundance, diversity, and productivity* (as decomposers and food chain base, among others) are ecological values to be preserved in a terrestrial ecosystem because they provide a mechanism for the physical and chemical breakdown of detritus for microbial decomposition (remineralization), which is a vital function.

5.2 ESTUARINE WETLAND AND AQUATIC ASSESSMENT ENDPOINTS

The estuarine wetland habitat for the Site extends over the majority of the North Area while the Intracoastal Waterway (i.e., aquatic habitat) is south of the Site. Wetlands are particularly important habitat because they often serve as a filter for water prior to it going into another water body. They are also important nurseries for fish, crab, and shrimp, and they act as natural detention areas to prevent flooding. The environmental value for these areas is related to their ability to support wetland plant communities, microbes/benthos/detritivores in the sediment, and wildlife. Based on the steps taken in the refinement (Section 2.0) and new information obtained about COPEC fate and transport and ecosystems at risk (Section 4.0), the following remains the assessment endpoint for the BERA (Table 2):

- *Benthos abundance, diversity, and productivity* are values to be preserved in estuarine ecosystems because these organisms provide a critical pathway for energy transfer from detritus and attached algae to other omnivorous organisms (e.g., polychaetes and crabs) and carnivorous organisms (e.g., black drum and sandpipers), as well as integrating and transferring the energy and nutrients from lower trophic levels to higher trophic levels. The most important service provided by benthic detritivores is the physical breakdown of organic detritus to facilitate microbial decomposition.

6.0 CONCEPTUAL SITE MODEL AND RISK QUESTIONS

6.1 CONCEPTUAL SITE MODEL

Preliminary Conceptual Site Models (CSMs) for the aquatic and terrestrial ecosystems were described in the SLERA. During problem formulation in the BERA, these CSMs have been updated to consider the results of the COPEC refinement (Section 2.0), expanded review of potential ecological effects of those COPECs (Section 3.0), and the more detailed fate and transport evaluation (Section 4.0). Updated CSMs based on these considerations are shown on Figures 9 and 10. These CSMs are discussed below.

The identification of potentially complete exposure pathways is performed to evaluate the exposure potential as well as the risk of effects on ecosystem components. In order for an exposure pathway to be considered complete, it must meet all of the following four criteria (EPA, 1997):

- A source of the contaminant must be present or must have been present in the past.
- A mechanism for transport of the contaminant from the source must be present.
- A potential point of contact between the receptor and the contaminant must be available.
- A route of exposure from the contact point to the receptor must be present.

Exposure pathways can only be considered complete if all of these criteria are met. If one or more of the criteria are not met, there is no mechanism for exposure of the receptor to the contaminant. The potentially complete and significant exposure pathways and receptors that match the current assessment endpoints are shown in the CSM for the terrestrial and estuarine wetland and aquatic ecosystems (Figures 9 and 10, respectively).

In general, biota can be exposed to chemical stressors through direct exposure to abiotic media or through ingestion of forage or prey that have accumulated contaminants. Exposure routes are the mechanisms by which a chemical may enter a receptor's body. Possible exposure routes include 1) absorption across external body surfaces such as cell membranes, skin, integument, or cuticle from the air, soil, water, or sediment; and 2) ingestion of food and incidental ingestion of soil, sediment, or water along with food. Absorption is especially important for plants and aquatic life.

The terrestrial ecosystem CSM (Figure 9) begins with historical releases of the COPECs from the former surface impoundments and operations areas in the North and South Areas. As noted in Section 2.2, there are no complete exposure pathways for the South Area soils. Soil became contaminated with the COPECs and contaminated soil was transported from its original location to other portions of the Site via the transport mechanisms of surface runoff and airborne suspension/deposition. The significant potential receptors (soil invertebrates) are then exposed to soils in their original location or otherwise via direct contact or ingestion of soil.

The aquatic ecosystem CSM (Figure 10) begins with historical releases of the COPECs from barge cleaning operations that impacted sediment in the barge slips of the Intracoastal Waterway and surface water and sediment in the North Area wetlands. These areas were impacted via the primary release mechanisms of direct discharge from past operations, surface runoff, and particulate dust/volatile emissions. Tidal flooding and rainfall events created secondary release mechanisms of resuspension/deposition, bioirrigation, and bioturbation, such that other areas of surface water and sediment became contaminated. The significant potential receptors (sediment and water-column invertebrates) are then exposed to the contaminated surface water and sediment in their original location or otherwise via direct contact or ingestion of surface water and sediment.

6.2 RISK QUESTIONS

As described in ecological risk assessment guidance (EPA, 1997), risk questions for the BERA are questions about the relationships among assessment endpoints and their predicted responses when exposed to contaminants. As such, the risk questions are based on the assessment endpoints and provide a basis for the ecological investigation study design developed in the BERA WP/SAP.

The overarching risk question to be evaluated in the BERA is whether Site-related contaminants are causing, or have the potential to cause, adverse effects on the invertebrates in North Area soils and on benthos and zooplankton of the wetlands area and the barge slips of the Intracoastal Waterway. For problem formulation, this overarching question is refined into a series of specific questions referencing specific COPECs and the assessment endpoint. Preliminary risk questions were developed for the Final SLERA (PBW, 2010a). Based on the information developed for this problem formulation, these risk questions were refined to the questions identified in Table 2

of this report. Testable hypotheses and measures of effect for these questions will be developed in the WP/SAP. The risk questions of concern for the end of the BERA Problem Formulation are the following:

- Does exposure to COPECs in soil adversely affect the abundance, diversity, productivity, and function of soil invertebrates?

- Does exposure to COPECs in sediment and surface water adversely affect the abundance, diversity, productivity, and function of sediment and water-column invertebrates?

7.0 SCIENTIFIC MANAGEMENT DECISION POINT

The final component of BERA problem formulation is an SMDP. The SMDP entails identification and agreement on the COPECs, assessment endpoints, exposure pathways, and risk questions that have been described in previous sections. As discussed above, the ecosystems potentially at risk for adverse effects are 1) localized areas of sediment within the Site barge slips (primarily due to PAHs); 2) localized wetland areas (primarily due to PAHs and pesticides), mainly northeast of the former surface impoundments and north of Marlin Avenue; and 3) a localized area of soils south of the former surface impoundments in the North Area.

The list of COPECs that will be addressed in the WP/SAP to obtain additional site-specific information is presented in Table 1.

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Tables

TABLE 1

COPECS AND MEDIA RECOMMENDED FOR FURTHER EVALUATION IN THE WORK PLAN FOR THE BASELINE ECOLOGICAL RISK ASSESSMENT

MEDIA	ASSESSMENT ENDPOINT	CHEMICAL OF POTENTIAL ECOLOGICAL CONCERN
North Area Soil	Direct Toxicity to Soil Invertebrates	4,4'-DDT Arochlor-1254 Barium Chromium Copper Zinc
Intracoastal Waterway Sediment	Direct Toxicity to Benthic Receptor	4,4'-DDT Acenaphthene Benzo(a)anthracene Chrysene Dibenz(a,h)anthracene Fluoranthene Fluorene Hexachlorobenzene Phenanthrene Pyrene LPAH HPAH Total PAH
Wetlands Sediment	Direct Toxicity to Benthic Receptor	2-Methylnaphthalene 4,4'-DDT Acenaphthene Acenaphthylene Anthracene Arsenic Benzo(a)anthracene Benzo(a)pyrene Benzo(g,h,i)perylene Chrysene Copper Dibenz(a,h)anthracene Endrin Aldehyde Endrin Ketone Fluoranthene Fluorene gamma-Chlordane Indeno(1,2,3-cd)pyrene Lead Nickel Phenanthrene Pyrene Zinc LPAH HPAH Total PAHs
Wetlands and Pond Surface Water	Direct Toxicity to Aquatic Invertebrates and Fish	Dissolved Copper Total Acrolein Dissolved silver
Pond Sediment	Direct Toxicity to Benthic Receptor	4,4'-DDT Zinc

Notes:
PAH - polynuclear aromatic hydrocarbon LPAH - low-molecular weight PAH HPAH - high-molecular weight PAH

TABLE 2
ASSESSMENT ENDPOINTS AND RISK QUESTIONS

Guild	Receptor of Potential Concern	Assessment Endpoint for BERA	Ecological Risk Questions
Invertebrates	Earthworm	Protection of soil invertebrate community from uptake and direct toxic effects on detritivore abundance, diversity, productivity from COPECs in soil.	Does exposure to COPECs in soil adversely affect the abundance, diversity, productivity, and function?
Benthos and zooplankton	Polychaetes	Protection of benthic and water-column invertebrate communities from uptake and direct toxic effects on abundance, diversity, and productivity from COPECs in sediment and surface water.	Does exposure to COPECs in sediment and surface water adversely affect the abundance, diversity, productivity, and function?
Vertebrate Fish	Fish Community	Protection of fish communities from uptake and direct toxic effects on abundance, diversity, and productivity from COPECs in sediment and surface water.	Does exposure to COPECs in surface water adversely affect the abundance, diversity, productivity, and function?

Figures

TEXAS

QUADRANGLE LOCATION

N

Scale in Feet

0 1000 2000

Source:
Base map taken from http://www.tnris.state.tx.us Freeport, Texas 7.5 min. U.S.G.S. quadrangle, 1974.

TITLE:

SITE LOCATION MAP

REPORT:	PROBLEM FORMULATION BASELINE ECOLOGICAL RISK ASSESSMENT		
SITE:	GULFCO MARINE MAINTENANCE FREEPORT, BRAZORIA COUNTY, TEXAS		
PROJECT: 41568745	DRAWN: ZGK/NAB	FIGURE: 1	
DATE: MAY, 2010	CHECKED: DL		

URS
10550 RICHMOND AVE., SUITE 155
HOUSTON, TEXAS 77042
PH: 713-914-6699
FAX: 713-914-8404

EXPLANATION

Gulfco Marine Maintenance Site Boundary (approximate)

Lot Boundary (approximate)

N

Approx. Scale in Feet

0 125 250

Source of photo: H-GAC, Texas aerial photograph, 2006.

TITLE:

SITE MAP

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	DRAWN:	FIGURE:
41568745	ZGK/NAB	2
DATE: MAY, 2010	CHECKED: DL	

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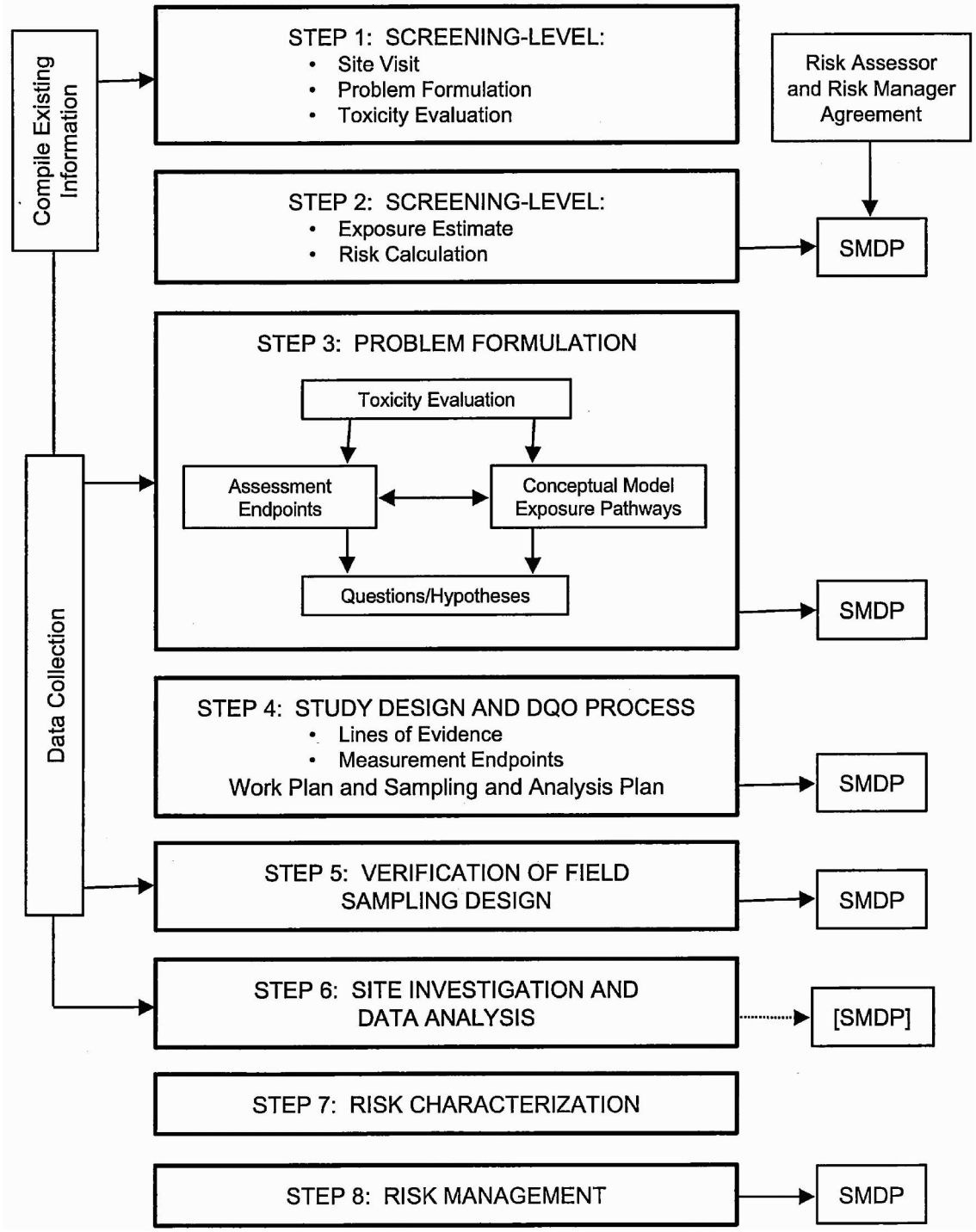

Source:
Exhibit I-2 from EPA Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments, 1997.

TITLE:

ECOLOGICAL RISK ASSESSMENT PROCESS

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	DRAWN:	FIGURE:
41568745	ZGK/NAB	3
DATE: MAY, 2010	CHECKED: DL	

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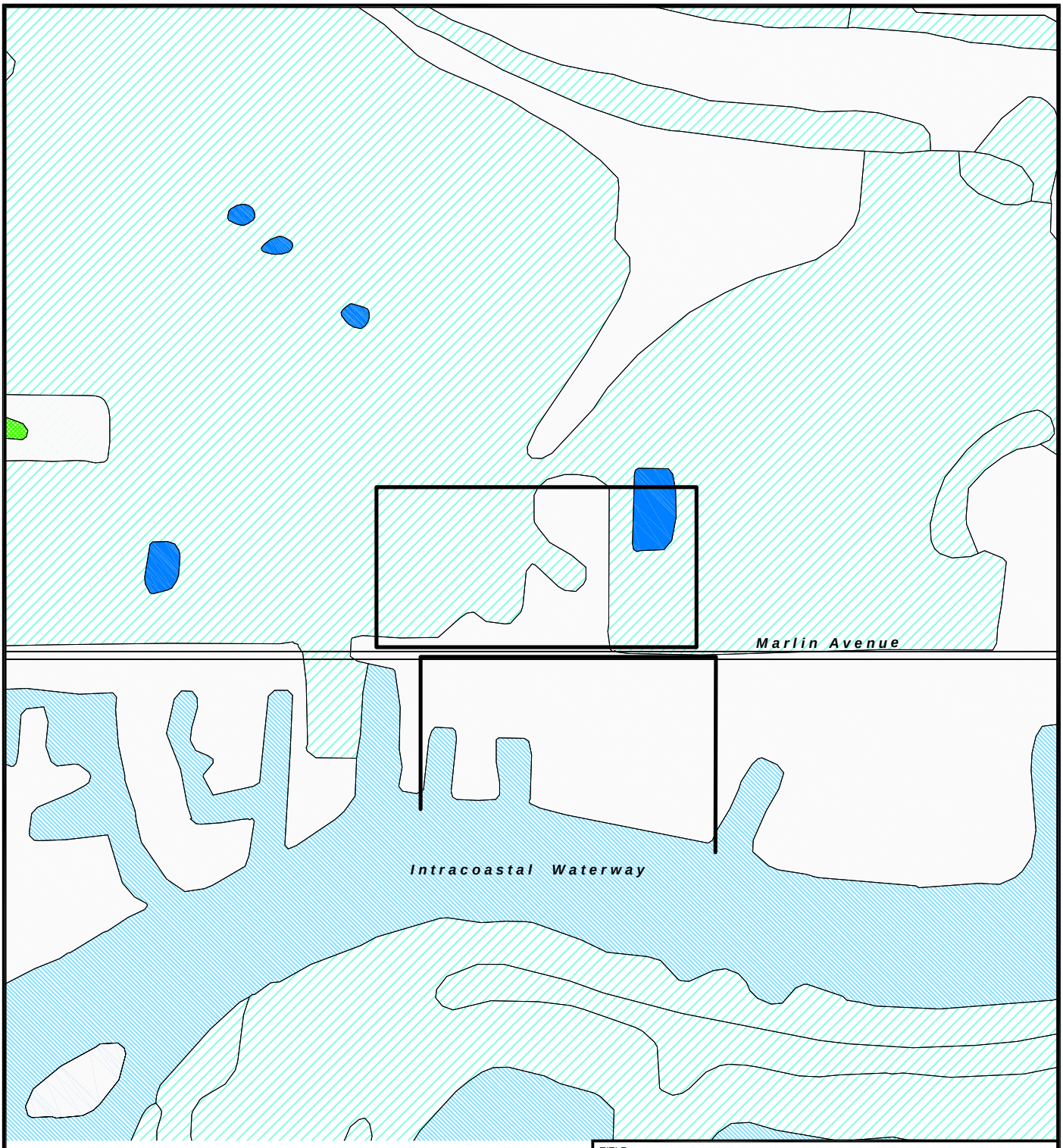

EXPLANATION

- Approx. Site Boundary
- Upland Area
- Estuarine and Marine Deepwater
- Estuarine and Marine Wetland
- Freshwater Emergent Wetland
- Freshwater Pond

N

Approx. Scale in Feet

0 300 600

Source:
U.S. Fish & Wildlife Service, Wetlands Online Mapper, 2008.

TITLE:

WETLAND MAP

REPORT: **PROBLEM FORMULATION BASELINE ECOLOGICAL RISK ASSESSMENT**

SITE: **GULFCO MARINE MAINTENANCE**
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	DRAWN:	FIGURE:
41568745	ZGK/NAB	4
DATE: MAY, 2010	CHECKED: DL	

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Depth (ft BGS)	Chemical of Interest	Hazard Quotient
1.5-2	4,4'-DDT	9
1.5-2	Aroclor-1254	3
1.5-2	Copper	2
1.5-2	Zinc	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Barium	1
0-0.5	Chromium	2
0-0.5	Zinc	47

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Zinc	2

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.8	Barium	1
0-0.8	Zinc	3

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Zinc	2
1-2	Barium	1
1-2	Zinc	2

EXPLANATION

- Gulfco Marine Maintenance Site Boundary (approximate)
- Shallow (0-2 ft) Soil Sample
- Shallow (0-2 ft) and Deep (4+ ft) Soil Sample
- Geotechnical Soil Boring

Notes:
1. BGS = below ground surface.
2. For sample concentration data, see SLERA Figures 7A through 7B.

* All Hazard Quotients for other receptors or compounds of concern were less than one. Hazard Quotients were based on No Observable Adverse Effects Level.

Approx. Scale in Feet

0 60 120

Source of photo: H-GAC, Texas aerial photograph, 2006.

TITLE:

HAZARD QUOTIENTS GREATER THAN ONE FOR SOIL INVERTEBRATES* - NORTH AREA SOIL

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT: 41568745 | DRAWN: ZGK/NAB | FIGURE: 5

DATE: JUNE, 2010 | CHECKED: DL

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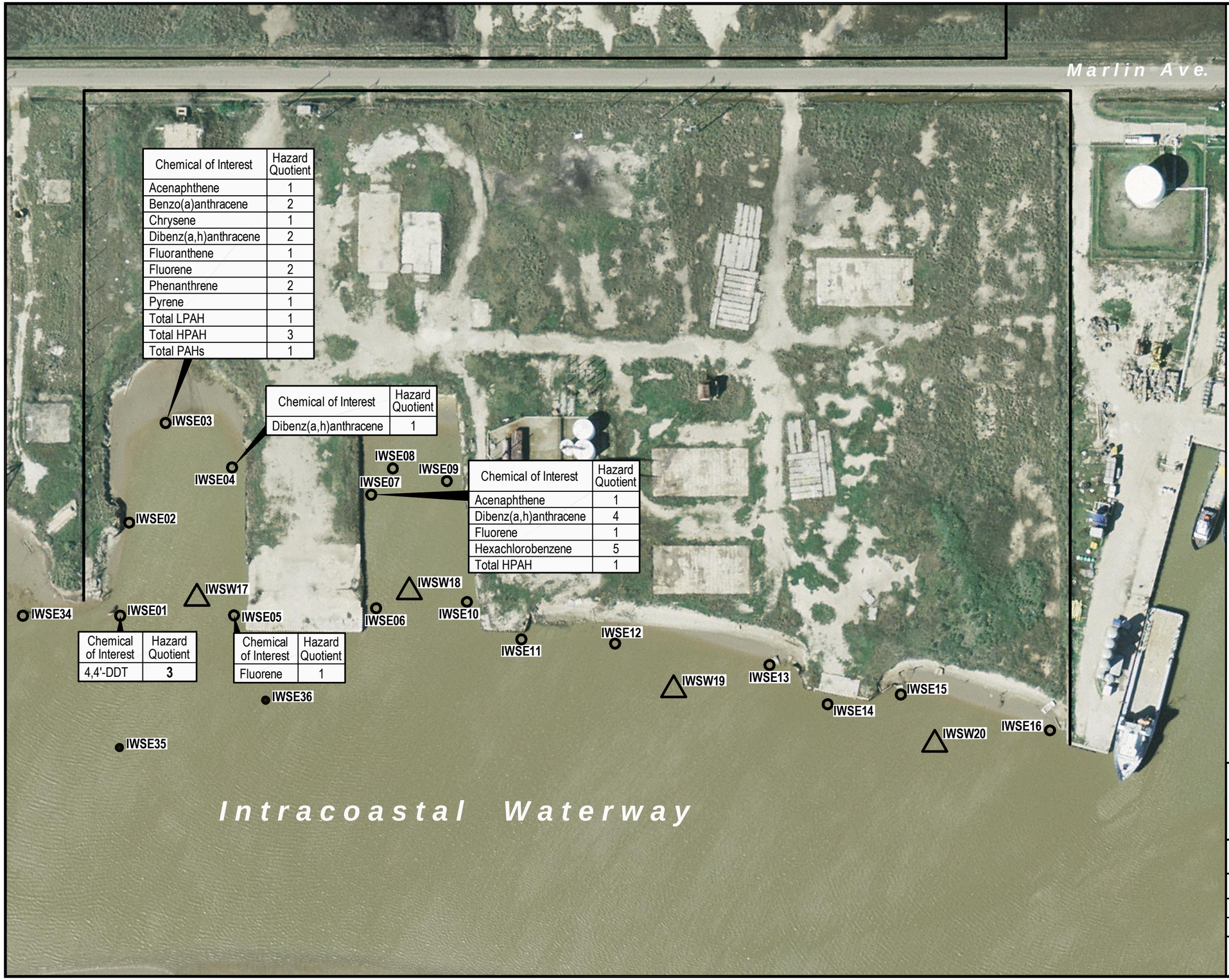

EXPLANATION

- Gulfco Marine Maintenance Site Boundary (approximate)
- Intracoastal Waterway Sediment Sample
- Intracoastal Waterway Surface Water Sample
- Attempted Intracoastal Waterway Sediment Sample (not enough sediment present to allow for sampling)

Notes:

1. For sample concentration data, see SLERA Figure 9.

* All Hazard Quotients for other receptors or compounds of concern were less than one. HQs for benthic receptors were based on the TCEQ sediment benchmarks.

Approx. Scale in Feet

0 60 120

Source of photo: H-GAC, Texas aerial photograph, 2006.

TITLE:

HAZARD QUOTIENTS GREATER THAN ONE FOR BENTHIC RECEPTORS*- INTRACOASTAL WATERWAY SEDIMENT

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	DRAWN:	FIGURE:
41568745	ZGK/NAB	6
DATE: JUNE, 2010	CHECKED: DL	

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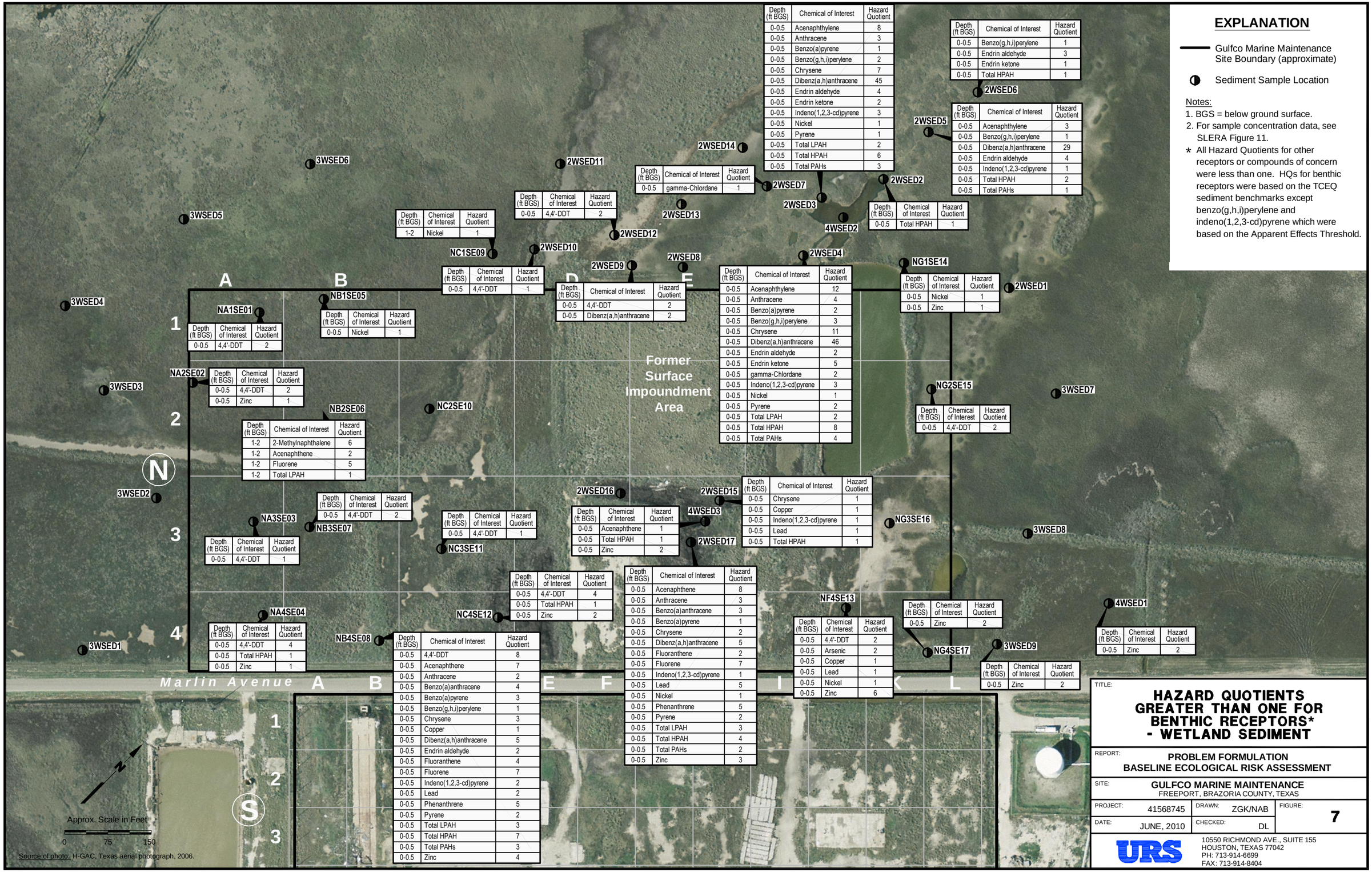

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	2

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Nickel	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
1-2	Nickel	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	2
0-0.5	Dibenz(a,h)anthracene	2

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Acenaphthylene	12
0-0.5	Anthracene	4
0-0.5	Benzo(a)pyrene	2
0-0.5	Benzo(g,h,i)perylene	3
0-0.5	Chrysene	11
0-0.5	Dibenz(a,h)anthracene	46
0-0.5	Endrin aldehyde	2
0-0.5	Endrin ketone	5
0-0.5	gamma-Chlordane	2
0-0.5	Indeno(1,2,3-cd)pyrene	3
0-0.5	Nickel	1
0-0.5	Pyrene	2
0-0.5	Total LPAH	2
0-0.5	Total HPAH	8
0-0.5	Total PAHs	4

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Nickel	1
0-0.5	Zinc	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	2

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
1-2	2-Methylnaphthalene	6
1-2	Acenaphthene	2
1-2	Fluorene	5
1-2	Total LPAH	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	2

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	4,4'-DDT	4
0-0.5	Total HPAH	1
0-0.5	Zinc	2

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Acenaphthene	8
0-0.5	Anthracene	3
0-0.5	Benzo(a)anthracene	3
0-0.5	Benzo(a)pyrene	1
0-0.5	Chrysene	2
0-0.5	Dibenz(a,h)anthracene	5
0-0.5	Fluoranthene	2
0-0.5	Fluorene	7
0-0.5	Indeno(1,2,3-cd)pyrene	1
0-0.5	Lead	5
0-0.5	Nickel	1
0-0.5	Phenanthrene	5
0-0.5	Pyrene	2
0-0.5	Total LPAH	3
0-0.5	Total HPAH	4
0-0.5	Total PAHs	2
0-0.5	Zinc	3

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Chrysene	1
0-0.5	Copper	1
0-0.5	Indeno(1,2,3-cd)pyrene	1
0-0.5	Lead	1
0-0.5	Total HPAH	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Acenaphthene	8
0-0.5	Anthracene	3
0-0.5	Benzo(a)pyrene	1
0-0.5	Benzo(g,h,i)perylene	2
0-0.5	Chrysene	7
0-0.5	Dibenz(a,h)anthracene	45
0-0.5	Endrin aldehyde	4
0-0.5	Endrin ketone	2
0-0.5	Indeno(1,2,3-cd)pyrene	3
0-0.5	Nickel	1
0-0.5	Pyrene	1
0-0.5	Total LPAH	2
0-0.5	Total HPAH	6
0-0.5	Total PAHs	3

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	gamma-Chlordane	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Total HPAH	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Benzo(g,h,i)perylene	1
0-0.5	Endrin aldehyde	3
0-0.5	Endrin ketone	1
0-0.5	Total HPAH	1

Depth (ft BGS)	Chemical of Interest	Hazard Quotient
0-0.5	Acenaphthylene	3
0-0.5	Benzo(g,h,i)perylene	1
0-0.5	Dibenz(a,h)anthracene	29
0-0.5	Endrin aldehyde	4
0-0.5	Indeno(1,2,3-cd)pyrene	1
0-0.5	Total HPAH	2
0-0.5	Total PAHs	1

EXPLANATION

Gulfco Marine Maintenance Site Boundary (approximate)

Sediment Sample Location

Notes:

1. BGS = below ground surface.
2. For sample concentration data, see SLERA Figure 11.

* All Hazard Quotients for other receptors or compounds of concern were less than one. HQs for benthic receptors were based on the TCEQ sediment benchmarks except benzo(g,h,i)perylene and indeno(1,2,3-cd)pyrene which were based on the Apparent Effects Threshold.

TITLE:

HAZARD QUOTIENTS GREATER THAN ONE FOR BENTHIC RECEPTORS* - WETLAND SEDIMENT

REPORT: PROBLEM FORMULATION BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT: 41568745 | DRAWN: ZGK/NAB | FIGURE: 7

DATE: JUNE, 2010 | CHECKED: DL

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Chemical of Interest	Hazard Quotient
ZINC	4

Chemical of Interest	Hazard Quotient
ZINC	5

Chemical of Interest	Hazard Quotient
4,4'-DDT	1
ZINC	7

EXPLANATION

Gulfco Marine Maintenance Site Boundary (approximate)

Pond Sediment Sample Location

Notes:

1. For sample concentration data, see SLERA Figure 12.

* All Hazard Quotients for other receptors or compounds of concern were less than one. HQs for benthic receptors were based on the TCEQ sediment benchmarks.

N

Approx. Scale in Feet

0 60 120

Source of photo: H-GAC, Texas aerial photograph, 2006.

TITLE:

HAZARD QUOTIENTS GREATER THAN ONE FOR BENTHIC RECEPTORS* - PONDS SEDIMENT

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	DRAWN:	FIGURE:
41568745	ZGK/NAB	8
DATE: JUNE, 2010	CHECKED: DL	

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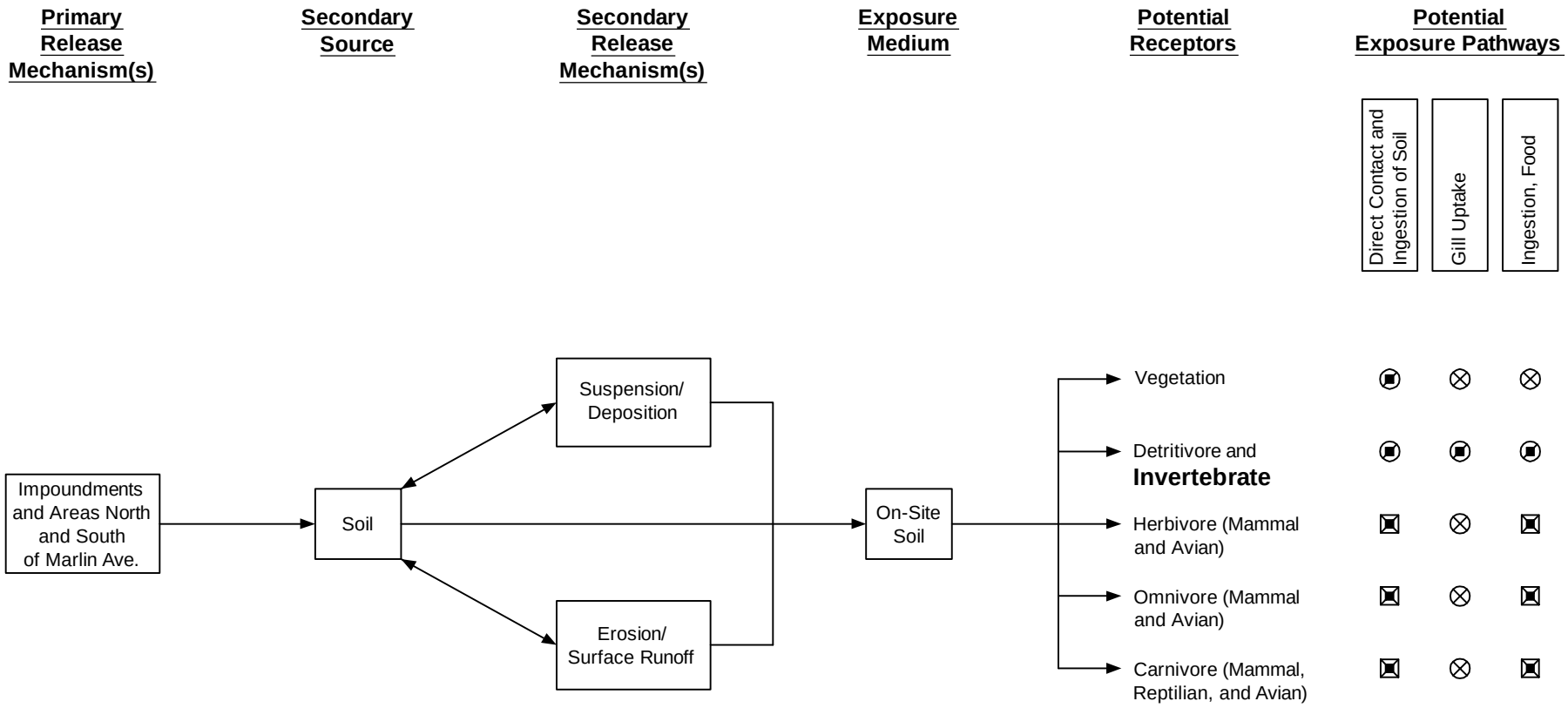

LEGEND

- ⊡ For South Area soils, pathway is mitigated by lack of complete exposure pathways. For North Area soils, pathway is potentially complete.
- ⊠ No unacceptable risk (Final SLERA conclusion)
- ■ Pathway is potentially complete
- ⊠ Pathway is incomplete
- ⊗ Pathway is not viable

Note:
Bolded receptors are those remaining for evaluation in the BERA after Problem Formulation refinement.

TITLE:

TERRESTRIAL ECOSYSTEM CONCEPTUAL SITE MODEL

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	DRAWN:	FIGURE:
41568745	ZGK/NAB	9
DATE: JUNE, 2010	CHECKED: DL	

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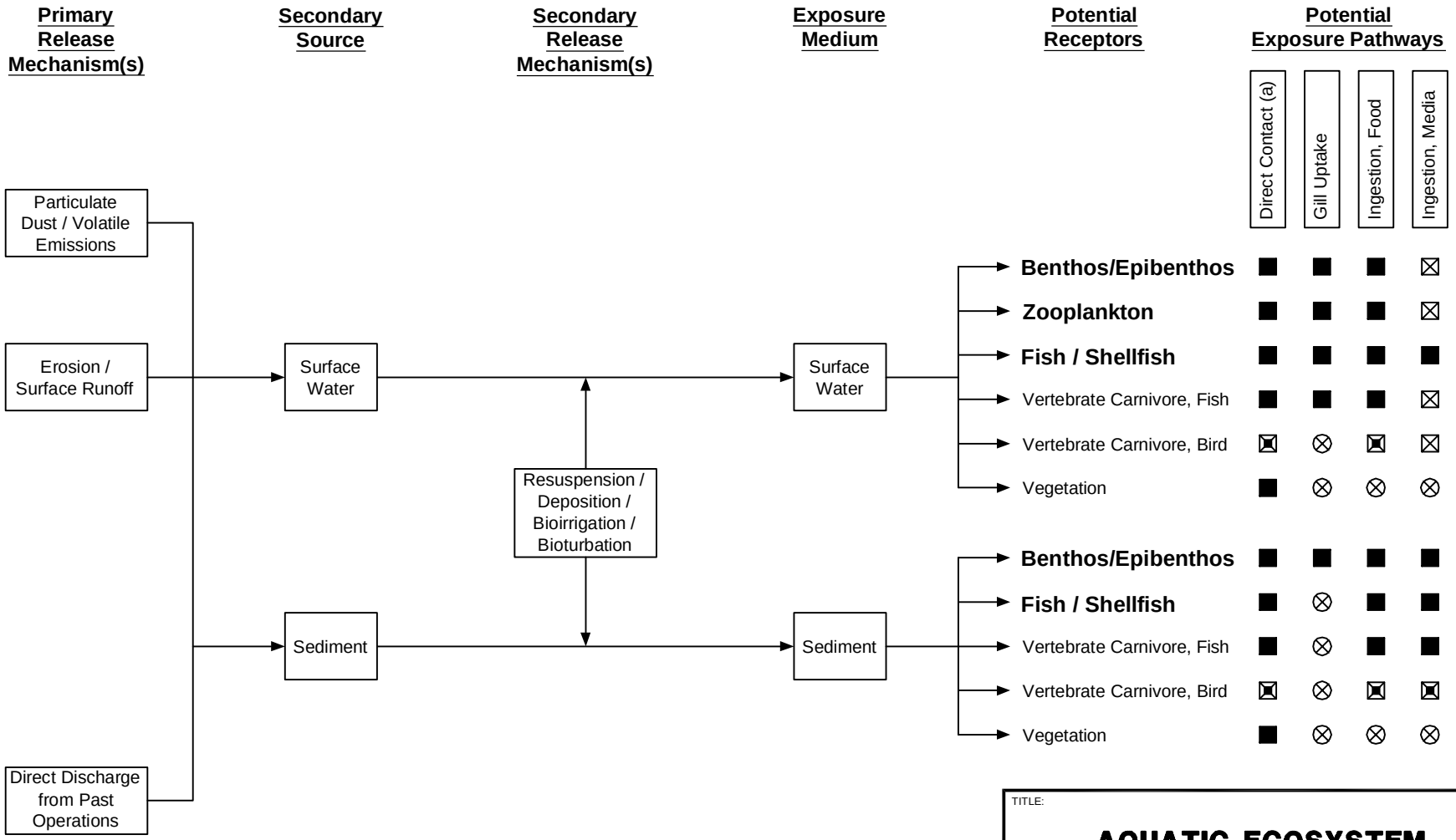

LEGEND

- ☒ No unacceptable risk (Final SLERA conclusion)
- ■ Pathway is potentially complete
- ☒ Pathway is incomplete
- ⊗ Pathway is not viable
- (a) Direct contact includes dermal absorption

Note:
Bolded receptors are those remaining for evaluation in the BERA after Problem Formulation refinement.

TITLE:

AQUATIC ECOSYSTEM CONCEPTUAL SITE MODEL

REPORT: PROBLEM FORMULATION
BASELINE ECOLOGICAL RISK ASSESSMENT

SITE: GULFCO MARINE MAINTENANCE
FREEPORT, BRAZORIA COUNTY, TEXAS

PROJECT:	41568745	DRAWN:	ZGK/NAB	FIGURE:	10
DATE:	JUNE, 2010	CHECKED:	DL		

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Appendix A

Table 29 (COPECs and Media Recommended for Further Evaluation in the Baseline Ecological Risk Assessment) from SLERA

TABLE 29
COPECS AND MEDIA RECOMMENDED FOR FURTHER EVALUATION IN THE BASELINE ECOLOGICAL RISK ASSESSMENT

MEDIA	ASSESSMENT ENDPOINT	CHEMICAL OF POTENTIAL ECOLOGICAL CONCERN
South Area Soil	Direct Toxicity to Soil Invertebrate	4,4'-DDD 4,4'-DDE 4,4'-DDT Aroclor-1254 Barium Chromium Copper Zinc Total HPAH
North Area Soil	Direct Toxicity to Soil Invertebrate	4,4'-DDT Aroclor-1254 Barium Chromium Copper Zinc
Intracoastal Waterway Sediment	Direct Toxicity to Benthic Receptor	4,4'-DDT Acenaphthene Benzo(a)anthracene Chrysene Dibenz(a,h)anthracene Fluoranthene Fluorene Hexachlorobenzene Phenanthrene Pyrene LPAH HPAH Total PAH
Wetlands Sediment	Direct Toxicity to Benthic Receptor	2-Methylnaphthalene 4,4'-DDT Acenaphthene Acenaphthylene Anthracene Arsenic Benzo(a)anthracene Benzo(a)pyrene Benzo(g,h,i)perylene Chrysene Copper Dibenz(a,h)anthracene Endrin Aldehyde Endrin Ketone Fluoranthene Fluorene gamma-Chlordane Indeno(1,2,3-cd)pyrene Lead Nickel Phenanthrene Pyrene Zinc LPAH HPAH Total PAHs
Wetlands Surface Water	Direct Toxicity to Aquatic Invertebrate	Acrolein Copper
Pond Sediment	Direct Toxicity to Benthic Receptor	4,4'-DDT Zinc
Pond Surface Water	Direct Toxicity to Aquatic Invertebrate	Silver

Notes:
PAH - polynuclear aromatic hydrocarbon
LPAH - low-molecular weight PAH
HPAH - high-molecular weight PAH

Appendix B

Environmental Fate/Transport and Toxicological Profiles

Acrolein

Environmental Fate and Transport

Sources

Acrolein is a colorless or yellow liquid with a disagreeable odor. It dissolves in water very easily and quickly changes to a vapor when heated. It also burns easily. Small amounts of acrolein can be formed and can enter the air when trees, tobacco, other plants, gasoline, and oil are burned. Acrolein is used as a pesticide to control algae, weeds, bacteria, and mollusks. It is also used to make other chemicals (http://www.atsdr.cdc.gov/tfacts124.html).

Fate and Transport

In the environment:

- Acrolein may be found in soil, water, or air.
- It breaks down fairly rapidly in the air (about half will disappear within 1 day) by reacting with other chemicals and sunlight.
- Acrolein evaporates rapidly from soil and water. (http://www.atsdr.cdc.gov/tfacts124.html).
- There were no studies identified on the bioavailabilty of acrolein in the environment (ATSDR, 2007)

Toxicological Profile

There is limited information on the toxicity to ecological receptors. No data were found regarding acrolein toxicity in plants. Acute toxicity values were developed after 4- hours of exposure to the rabbits. The acute oral toxicity of acrolein on rabbits was found to be 7 mg/kg. The acute dermal toxicity of acrolein on rabbits was found to be 200 mg/kg (Science Lab.com, 2010).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological profile for Acrolein. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Science Lab.com 2010 On-line Material Safety Data Sheet.

ARSENIC

Environmental Fate and Transport

Sources

Arsenic is an element that occurs naturally in a variety of sulfidic ores. Arsenic can be released to the environment from natural sources (e.g., volcanoes, erosion from mineral deposits), but releases from human activities (e.g., metal smelting, chemical production and use, coal combustion, waste disposal) can lead to substantial environmental contamination. Most anthropogenic releases of arsenic are to land or soil, primarily in the form of pesticides or solid wastes; however, substantial amounts are also released to air and water. Arsenic released to land is relatively immobile, due to binding to soil particles. Rainwater or snowmelt may leach soluble forms into surface water or groundwater and soil microorganisms may reduce a small amount to volatile forms (arsines). Arsenic dissolved in water can undergo either reduction or oxidation, depending on conditions. Poorly soluble forms tend to adsorb to organic material in sediments or soils, while the soluble species tend to move with water (Agency for Toxic Substances and Disease Registry [ATSDR], 1993).

Transport and Fate

Arsenic is both reactive and mobile and can cycle extensively through both biotic and abiotic components of local aquatic and terrestrial systems. It can undergo a variety of chemical and biochemical transformations, such as oxidation, reduction, methylation, and demethylation (Environment Canada, 1993). Most arsenic in the environment exists in soil or rock. While it can be transported by wind and water erosion and runoff, most arsenic in water is adsorbed to soil or sediment (ATSDR, 1993).

Transport and partitioning of arsenic in water depends upon the chemical form (oxidation and ionic state). Sediment-bound arsenic may be released back into the water by chemical or biological inter-conversions of arsenic species (ATSDR, 1993). Arsenate (As+5) is the main species found in oxidizing environments. Reducing conditions, such as anoxic sediments, favor the more hazardous form, arsenite (As^{+3}) (Moore et al., 1990).

Soluble arsenic in surface water may be carried long distances in rivers. The ultimate sink for arsenic is in ocean sediment. Arsenic tends to adsorb to soil and leaching usually results in transportation over only short distances. In highly contaminated areas, ground water normally will have higher concentrations of arsenic than are found in associated surface water (Irwin et al, July 1997).

Speciation and Bioavailability

The toxicity of inorganic compounds containing arsenic depends on the valence or oxidation state of the arsenic ($^{(-3,\ +3,\ or\ +5}$), as well as on the physical and chemical properties of the compound in which it occurs. Trivalent (As^{+3}) compounds such as arsenic trioxide (As_2O_3), arsenic trisulfide (As_2S_3), and sodium arsenite ($NaAsO_2$), are generally more toxic than pentavalent (As^{+5}) compounds such as arsenic pentoxide (As_2O_5), sodium arsenate (Na_2HAsO_4), and calcium arsenate ($Ca_3(AsO_4)_2$). Trivalent

arsenic interacts with sulfhydryl groups of proteins and enzymes; pentavalent arsenic substitutes for phosphate groups important in oxidative phosphorylation (Squibb and Fowler, 1983).

The two primary forms of arsenic in water are trivalent arsenic and pentavalent arsenic. The relative toxicity of the trivalent and pentavalent forms may also be affected by factors such as the water solubility of the compound. Soluble inorganic arsenate ($^{+5}$ oxidative state) predominates under normal conditions since it is thermodynamically more stable in water than arsenite ($^{+3}$ oxidative state). The more water-soluble arsenic compounds are generally more toxic. Arsenic toxicity in water is not governed by hardness. Arsenic is one of the most toxic elements to fish (Irwin et al., July 1997).

Bioconcentration of arsenic in aquatic organisms is primarily in algae and lower invertebrates. Bioconcentration factors measured in freshwater invertebrates and fish for several arsenic compounds ranged from 0 to 17. The potential for bioaccumulation or bioconcentration of arsenic is moderate for mammals, birds, fish, mosses, lichens, and algae. The potential is considered high to very high for mollusks, crustaceans, lower animals, and higher plants (Jenkins, 1981). Biomagnification in aquatic food chains does not appear to be significant, although some fish and invertebrates contain high levels of arsenic compounds. Terrestrial plants may accumulate arsenic by root uptake from the soil or by absorption of airborne arsenic deposited on the leaves, and certain species may accumulate substantial levels (ATSDR, 1993).

Toxicological Profile

Summary

Arsenic is ubiquitous in nature and is a required nutrient in several animal species. Organic arsenic compounds are used as animal feed additives in swine, chickens, and turkeys to increase the rate of weight gain, to control swine dysentery, to act as an anti-histomonad in chickens, and to increase egg production in fowl. There is no evidence that arsenic is essential for plant growth (Irwin et al., July 1997).

Plants

Plants are generally more sensitive to arsenic than animals. Minimum soil concentrations causing phytotoxicity are 15 to 50 mg/kg dry weight. More sensitive plants, like rice, can be affected at arsenic concentrations above 7 mg/kg, or more sensitive life stage, like newly established vegetation may eventually be killed at 17 mg/kg arsenic in soil (Irwin et al, July 1997). Depression in crop yields for most plants was seen at soil total arsenic concentrations of 25 to 85 mg/kg. The current Eco-SSL for protection of plants is 18 mg/kg (United States Environmental Protection Agency [EPA], March 2005). The pH, moisture, and concentration of phosphorus, aluminum, iron and calcium affects the availability of arsenic in soil to uptake in plants (Eisler, 1988). The edible portions of plants grown on contaminated sources seldom accumulate dangerous levels of arsenic because the plant will be killed before the arsenic the plant can assimilate dangerous levels in the edible portions and because phosphorus competes with arsenic to gain entry

into plants (Nriagu and Azcue, 1990).

Phytotoxic actions of inorganic and organic arsenic are different. The primary mode of action for arsenite in plants is inhibition of light activation as indicated by wilting. Organoarsenical herbicide phytotoxicity is characterized by chlorosis, cessation of growth, gradual browning, dehydration, and death (Eisler, 1988).

Invertebrates

No information was found for soil or sediment concentrations versus effects in invertebrates (Eisler, 1988; Irwin et al., July 1997). There is not enough information for the development of an invertebrate Eco-SSL for arsenic (EPA, March 2005).

Fish

Fin fish (carp, eels, perch, pike) exposed to 1-2 ppm arsenic for 2-3 days may show some or all of the following signs of toxicity: hemorrhagic spheres on gills, fatty infiltration of the liver, cellular necrosis of heart and liver tissues, and cellular disruption in the ovaries. Young fish were more susceptible to arsenic toxicity than were adults: threshold acute toxic levels in tissue were 1.3 ppm and 5 ppm respectively in young and mature bluegills. Behavioral effects were observed in fish at low arsenic levels: 0.1 ppm Na3AsO4 produced 30% impairment of conditioned responses in goldfish. Increasing water temperature promoted greater arsenic uptake by fish and also increased the intrinsic toxicity of a given amount of absorbed arsenic (NRC Canada, 1978).

A chronic study using juvenile rainbow trout, fed semi-purified diets containing graded levels of disodium arsenate heptahydrate (DSA) for 12-24 weeks under standard laboratory conditions, found that the maximum acceptable toxicant concentration for DSA was between 13 and 33 micrograms As/g diet or 0.281-0.525 mg As/kg body weight/day. The most sensitive and reliable indicator of chronic dietary DSA toxicity in rainbow trout was chronic inflammation of the gallbladder wall. Chronic inflammatory changes in the sub-epithelial tissues of the gallbladder wall were evident in 71% of rainbow trout exposed to 33 micrograms As/g diet for 24 weeks, and 100% of rainbow trout exposed to 65 micrograms As/g diet for 24 weeks or 49 micrograms As/g diet for 12 weeks. No fish exposed to 13 micrograms As/g diet or less for up to 24 weeks showed any demonstrable gallbladder lesions or any other ill effect of arsenic exposure. Other signs of chronic dietary DSA toxicity to rainbow trout included decreased growth rate, mild to moderate anemia, and, at higher levels of exposure, active feed refusal leading to decreased feed consumption. Mild nephrocalcinosis was noted in one experiment where kidney arsenic residues exceeded 14 micrograms As/g tissue dry weight (Cockell et al., 1991).

Terrestrial Vertebrates

Arsenic toxicity is well documented in terrestrial vertebrates and the degree and effects differ, depending upon exposure pathway, duration, and animal species tested. The current Eco-SSLs for protection of birds and mammals are 43 mg/kg and 46 mg/kg, respectively (EPA, March 2005).

Mammals

In animals, acute oral exposures can cause gastrointestinal and neurological effects (Heywood and Sortwell, 1979). Oral LD_{50} values range from about 10 to 300 mg/kg (ASTDR, 1989; U.S. Air Force, 1990). Low subchronic doses can result in immunosuppression (Blakely et al., 1980) and hepato-renal effects (Brown et al., 1976; Woods and Fowler, 1977 and 1978; Fowler and Woods, 1979; Fowler et al., 1979). Chronic exposures have also resulted in mild hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis (Baroni et al., 1963; Byron et al., 1967). Reduction in litter size, high male/female birth ratios, and fetotoxicity without significant fetal abnormalities occur following oral exposures (Schroeder and Mitchener, 1971; Hood et al., 1977; Baxley et al., 1981); however, parenteral dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities (Ferm and Carpenter, 1968; Hood and Bishop, 1972; Beaudoin, 1974; Burk and Beaudoin, 1977). LD_{50} values for inorganic arsenic compounds in laboratory animals range from about 10 to 300 mg/kg (ASTDR, 1989; U.S. Air Force, 1990).

Birds

The signs of inorganic trivalent arsenite poisoning in birds (mallard, quail, pheasant) include ataxia, goose-stepping ataxia, asthenia, slowness, jerkiness, falling, hyporeactivity, fluffed feathers, ptosis, huddled position, unkempt appearance, loss of righting reflex, immobility, and titanic seizures. Signs appear as soon as 1 hour and mortalities usually after exposure; remission takes up to 1 month (Moore et al., 1990).

Nestling northern bobwhites, mockingbirds, American robins and other songbirds fed grasshoppers containing a total of 40 mg arsenic (as arsenic trioxide) did not show deleterious effects. Brown-headed cowbirds have an oral LD-50 (11 day) value of 100 mg of copper acetoarsenite/kg diet. California quail has a single oral dose LD-50 value of 48 mg sodium arsenite/kg body weight, and chicken with 33 and turkey with 17 mg/kg body weight of 3-mitro-4-hydroxy phenylarsonic acid (Eisler, 1988). Chickens rapidly excrete arsenicals with only 2% dietary sodium arsenite remaining after 60 hours (Eisler, 1988).

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BARIUM

Environmental Fate and Transport

Sources

Barium occurs in nature in a combined state, the principal forms being barite (barium sulfate) and witherite (barium carbonate). Barium is also present in small quantities in igneous rocks such as feldspar and micas and may also be found as a natural component of fossil fuels. The production and use of various barium compounds in pyrotechnic devices, ceramics, paints, enamels, optical glasses, and as a getter to remove traces of gas from vacuum and television tubes may result in its release to the environment though various waste streams. Barium is emitted into the environment mainly by the industrial processes involved in mining, refining and production of barium and barium-based chemicals and as result of combustion of coal and oil (EPA November, 2003).

Barium metal does not occur freely in nature (Kirk-Othmer Encyclopedia of Chemical Technology, 1978-1984). Seawater contains about 0.03 ppm barium (Venugopal and Luckey, 1978). Background levels in natural soils range from 100-700 mg/kg dw barium (EPA, November 2003). The average concentration of barium in air was reported to be 5 ng/m^3 (0-1500 ng/m^3) in 18 U.S. cities (Friberg et al., 1986).

Fate and Transport

Barium is not very mobile in most soils. The rate of transportation of barium in soils is dependent on soil characteristics. Soil properties that influence the transportation of barium to groundwaters are cation exchange capacity and calcium carbonate (CaCO3) content. In soils with a high cation exchange capacity (e.g., fine textured mineral soils or soils with high organic matter content), barium mobility will be limited by adsorption (Lagas et al., 1984). High calcium carbonate content limits mobility by precipitation of the element as barium carbonate. In soils, barium will also precipitate as barium sulfate in the presence of sulfate ions (Bodek et al., 1988, and Lagas et al., 1984).

The solubility and mobility of barium is greater in sandy soil increasing with decreased pH and decreased organic matter. Barium can react with metal oxides and hydroxides in soils, thus limiting its mobility and increasing adsorption. Barium mobility decreased in soils with high sulfate and calcium carbonate content (EPA, November 2003).

In aquatic media, barium compounds are likely to precipitate out of solution as barium sulfate (BaSO4) or barium carbonate (BaCO3). Waterborne barium may also adsorb to suspended particulate matter (EPA, 1984, Bodek et al., 1988, and Lagas et al., 1984). Sedimentation removes a large portion of barium compounds that are suspended in surface waters (Benes et al., 1983).

Appreciable quantities of barium sulfate or carbonate precipitate may occur in aquatic environments. This is because natural waters usually contain sulfate or carbonate concentrations that are sufficient to react with barium ion to form barium sulfate or carbonate, which precipitates from solution (NAS, 1977). In natural waters at pH levels of

9.3 or below, barium ion will react to form barium sulfate (Bodek et al., 1988). At pH above 9.3 formation of barium carbonate is favored.

Speciation and Bioavailability

Barite is a common mineral found in medium and low temperature hydrothermal veins associated with lead, silver, and antimony sulfides and in replacement veins, cavernous limestone formations, and dolomites. Witherite occurs in low temperature hydrothermal veins and is associated with barite and galena (Mottana, 1978).

Barium does not bioaccumulate in the aquatic environment, and concentrations in higher aquatic food web species rarely exceed 10 mg/kg (Moore, 1991).

Toxicological Profile

Aquatic Organisms and Plants

Barium compounds have low toxicity to aquatic organisms and plants (EPA, June 1993 and January 1997). The low toxicity of barium compounds to aquatic species is attributable to the presence of sulfate in waters; barium ion liberated from a barium compound reacts with sulfate to form barium sulfate, which precipitates from solution.

Soil Invertebrates

Very limited toxicity data are available describing effects of barium on soil invertebrates. Soil invertebrates can survive in pure barium sulfate. The current Eco-Soil Screening Level (SSL) for protection of soil invertebrates is 330 mg/kg (EPA, February 2005).

Terrestrial Plants

Very limited toxicity data are available describing effects of barium on terrestrial plants. Plants can survive in pure barium sulfate, but there is some growth reduction. Chaudhry et al. (1977) reported for reduction in plant weight a LOEC of 2000 mg/kg and NOEC of 1000 mg/kg for bush beans and a LOEC of 500 mg/kg for barley. There is not enough information for the development of a plant Eco-SSL for barium (EPA, February 2005).

Terrestrial Vertebrates

Mammals

Review of the available toxicity data for barium compounds identified kidney toxicity as the toxicological endpoint of concern (NTP, 1993). Based on the renal toxicity of barium chloride dihydrate to mice in drinking water for 2 years, the incidence of nephropathy was significantly increased in mice of both genders that received 2,500 mg/L. The LOAEL was 160 mg/kg/day for male mice and 200 mg/kg/day for female mice. The No Observed Adverse Effect Level (NOAEL) was 75 mg/kg/day for male mice and 90 mg/kg/day for female mice. Thirty percent of male rats receiving 4,000 mg/L barium chloride dehydrate for 13 weeks died, and the other rats had a significant decrease in motor activity at this concentration (equal to 200 mg/kg/day). In mating trials, no anatomical effects on offspring of rats or mice were noted. No effects were noted on reproductive indices. Based on the

mortality and renal toxicity at 4,000 mg/L in both rats and mice, the NOAEL was 70 mg/kg/day in rats and 165 mg/kg/day in mice (NTP, 1993).

The acute oral lethality of barium in animals has been well documented. There is a wide variability in the lethal dose of barium among species and age, as well as between strains of the same species. Nevertheless, the acute lethality of various barium salts is a function of their solubility in water or acid. In rats, acute oral toxicities of barium chloride, fluoride, nitrate and acetate have median lethal dose (LD50) values of 118, 250, 355 and 921 mg barium/kg, respectively. (WHO, 1991). The current Eco-Soil Screening Level (SSL) for protection of mammals is 2,000 mg/kg (EPA, February 2005).

Birds

Very limited toxicity data are available describing effects of barium on soil invertebrates. Only one study was found. In one-day old chicks exposed to barium hydroxide for 4 weeks, the oral LOAEL was 41.7 mg/kg/day and the oral NOAEL was 20.8 mg/kg/day (Johnson et al., 1960). There is not enough information for the development of an avian Eco-SSL for antimony (EPA, February 2005).

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CHLORDANE

Environmental Fate and Transport

Sources

Chlordane was used as a pesticide on lawns, gardens, and crops before 1978. From 1983 through 1988, the only approved use for chlordane was termite control. After 1988, EPA banned the use of chlordane entirely after learning of the cancer risk associated with chlordane, the risk of human exposure, its ability to build up in body fat, its persistence in the environment and its threat to wildlife.

Transport and Fate

Chlordane attaches strongly to the particles in the upper soil layer. It is unlikely to reach groundwater. If chlordane breaks down in soils, it is very slow. The break down is slowest in clay-like soils or soils with high amounts of organic matter. Most chlordane evaporates from the soil, which is fastest in sandy soils. Persistence is greater in heavy, clayey or organic soil than in sandy soil. Chlordane is known to remain in some soils for over 20 years. Most chlordane is lost from soil by evaporation. Half of the chlordane applied to the soil surface may evaporate in 2 to 3 days (ATSDR 1994).

Chlordane attaches strongly to sediment once it is in the water. Chlordane does not dissolve in water. It is unknown if chlordane breaks down in water and sediment. However, the half-life of chlordane in surface water is comparatively short, less than 1 day, because of evaporation. Chlordane reacts with light and some chemicals in the atmosphere. However, it is persistent in that media, as well.

Speciation and Bioavailability

Chlordane is not a simple chemical, but is a mixture of many related chemicals, of which 10 are major components. Some of the major components are trans-chlordane, cis-chlordane, beta-chlordane, heptachlor, and trans-nonachlor. There is also gamma- and alpha-chlordane.

Chlordane is readily absorbed by warm-blooded animals through skin, diet, and inhalation. It is still found in the fat of fish, birds, mammals and almost all humans (ATSDR 1994). Food-chain biomagnification is usually low, except in marine mammals. In most mammals, the metabolite oxychlordane has proven to be much more toxic and persistent than the parent chemical (Eisler July 1990). Heptachlor epoxide and oxychlordane, metabolites, originate from biological and physical breakdown of chlordanes in the environment, or from metabolism after ingestion. Heptachlor can result from the breakdown of cis- and trans-chlordane, eventually oxidizing to heptachlor expoxide; oxychlordane can result form the breakdown of heptachlor, cis-chlordane, trans-chlordane, or trans-nonachlor (Blus et al. 1983).

Chlordane will bioconcentrate in both marine, bioconcentration factor 3,000 - 12,000 (Zaroogian et al.1985), and fresh water species, bioconcentration factor of 18,500 shown in rainbow trout (Oliver and Niimi 1985). Muir et al, 1988, studied the biomagnification

of chlordane-related compounds in three trophic levels of the Arctic marine food chain. The biomagnification from fish to seal (male/female) was 7.3/4.7 and that between seal (male/female) and bear was 6.6/9.5 resulting in an overall fish to bear biomagnification factor of 44.2 (Muir et al, 1988).

Toxicological Profile

Summary

Soil Invertebrates

Chlordane had been applied extensively to control pestiferous soil invertebrates (grubs, ants, snails, and termites) at rates between 0.6 and 2.24 kg/ha; within this range sensitive nontarget species, especially earthworms, were adversely affected (Eisler July 1990).

Fish and Aquatic Invertebrates

Many species of fish and aquatic invertebrates are adversely affected at concentrations in water between 0.2 and 3.0 µg/L technical chlordane (Eisler July 1990). Effects included a reduction in survival, immobilization, impaired reproduction, histopathology, and elevated chlordane accumulations (Eisler July 1990). Cis-chlordane, when compared to trans-chlordane, was more toxic, preferentially stored, and concentrated to a greater degree. In aquatic organisms, cis-chlordane photoisomers were frequently more toxic than the parent form. Oxychlordane was not a major metabolite in aquatic organisms (Eisler July 1990). Sediment concentration of 5.8 mg/kg was the LC_{50} for sandworms (McLeese et al. 1982).

Amphibians and Reptiles

Skinks, frogs, and toads have all been reported killed by application of chlordane for termite control (Eisler July 1990). For tadpoles of the common toad (*Bufo bufo*) a 48-hr LC_{50} is 2 mg/L (WHO 1984).

Terrestrial Vertebrates

Mammals

Chlordane is a nerve stimulant, at low chronic doses it produces hyperexcitability and lack of coordination in animals, and at high acute doses causes tremors and convulsions (Eisler July 1990). The physiological target sites are in nerve and muscle membranes, presumably on proteins and phospholipids; the ultimate effect is axonic with membrane disruption, resulting in spasmic muscle twitching and death (Greenhalgh 1986). The liver is another target organ (Klaassen et al. 1986).

Metabolism of technical chlordane by mammals results primarily in oxychlordane, which is about 20 times more toxic than the parent compound and the most persistent metabolite stored in fatty tissue (Eisler July 1990). Acute oral LD_{50} values for technical chlordane

and sensitive mammals ranged between 25 and 50 mg/kg-BW. Growth and fertility has also reported as affected by ingestion route of exposure.

Birds

Signs of chlordane toxicity in birds include sluggishness, drooped eyelids, fluffed feathers, low crouching on perch, reduced food intake, and weight loss. Later, afflicted birds rest on their breasts, wings spread, quivering and panting, back arched, neck arched over the back, and convulsing (Stickel et al. 1983).

Sensitive bird species had reduced survival on diets containing 1.5 mg chlordane per kg diet, or after a single oral dose as low as 14.1 mg chlordane/kg-body weight. Oxychlordane was the most persistent metabolite in avian brain tissue. Reproduction impairment was reported for waterfowl from treated marshes (Eisler July 1990).

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CHROMIUM

Environmental Fate and Transport

Sources

Chromium is a naturally occurring element found in rocks, plants and animals, soil, and volcanic dust and gases as Chromium (III). Other forms, such as Chromium (O) and Chromium (VI) are produced commercially. Chromium compounds of sodium, potassium, and aluminum are also produced industrially, principally using the forms Chromium (III) and Chromium (VI). These products are used for chrome plating, in the manufacture of dyes, leather (as a tanning agent), wood preservatives, in toner for copying machines, and in textiles. Chromium is a steel-gray metal that can be highly polished. It has a molecular weight of 52. It melts at 1857^{o}C and boils at 2670^{o}C.

Fate and Transport

Chromium release to the atmosphere occurs primarily from industry and fuel combustion. Natural releases result form continental dust and volcanic dust and gases (Fisbein, 1981; Towill et al., 1978). The predominant form of atmospheric chromium is particulate, and transport and deposition are determined primarily by particle size and density (Cary, 1982). Chromium is reduced by other elements and compounds present in air; however, removal of chromium from the atmosphere is accomplished through wet and dry deposition of the materials to soil and surface water (Schroeder et al., 1987).

Excessive chromium in surface and ground waters are generated by waste water from industry and deposition of airborne chromium. In water, oxidation and reduction reactions occur, generally at slow rates.

Disposal of commercial chromium-containing products, industrial disposal, agricultural wastes, animal wastes, and atmospheric deposition account for the presence of unnaturally high levels of chromium in soil. Most soil-bound chromium is in an insoluble form and not mobile; however, rainwater and anaerobic biodegradation of plant materials tend to increase mobility, particularly in acidic conditions (Stackhouse and Benson, 1989). Mobile chromium may be transported to surface water, ground water, or the atmosphere.

Speciation and Bioavailability

Chromium is not expected to bioaccumulate in aquatic organisms, based on the bioconcentration factors (BCF) for Chromium (III) and Chromium (VI) in oyster, fish, mussel, and clam (United States Environmental Protection Agency [EPA], 1980 and 1984; Fisbein, 1981).

Toxicological Profile

Plants

Chromium concentrations in plants grown in chromium-contaminated soil have been shown to have elevated chromium levels, however, most of the chromium remained in the plants'

root structures (Cary, 1982). There is not enough information for the development of a plant Eco-Soil Screening Level (SSL) for chromium (EPA, March 2005).

For freshwater plants, Suter and Tsao (1996) reported the lowest chronic screening value for chromium III and chromium VI as 397 µg/L and 2 µg/L, respectively. The toxicity threshold for diatoms was reported as 0.03 mg/kg for chromium VI (National Research Council, 1976). The chromium VI concentration of 62 µg/L inhibited the growth of *Selenastrum capricornutum* (green alga) (EPA, 1984).

Invertebrates

Abbasi and Soni (1983) exposed the earthworm *Octochaetus pattoni* to Cr(VI) added to a mixture of soil and manure as $K_2Cr_2O_7$ for 60 days to assess the effect on survival and reproduction. Survival was the most sensitive measure with a 75% decrease resulting from 2 mg/kg Cr, the lowest concentration tested. The number of cocoons produced was not diminished until the concentration reached 20 mg/kg Cr (highest concentration tested); the number of juveniles produced was not affected.

Van Gestel et al. (1993) found growth of *Eisenia andrei* to be more sensitive to Cr than reproduction. Cr(III) was added as chromic nitrate to soil; a concentration of 32 mg/kg Cr reduced growth by 30% while cocoons/worm/week, percent fertile cocoons, and juveniles/worm/week were reduced by 28, 22, and 51%, respectively, by 100 mg/kg Cr.

Molnar et al. (1989) examined the effects of Cr(III) and Cr(VI) on growth and reproduction of *Eisenia fetida* in an undefined substrate. Chromium (VI) was added as $K_2Cr_2O_7$ and Cr(III) as $KCr(SO_4)_2$. Reproduction after 8 weeks was the measure most sensitive to Cr(III) with a 55% decrease in the number of cocoons and hatchlings at 625 mg/kg Cr(III). The authors indicated that reproduction was also sensitive to Cr(VI) but no data was given. After 2 weeks, mass gain of juveniles was decteased 34% by 2,500 mg/kg Cr(III) (625 mg/kg had no effect) and 43% by 625 mg/kg Cr(VI) (lowest concentration tested). After 4 weeks, mass gain of juveniles was decreased by 39% by 2,500 mg/kg Cr(III) (625 mg/kg had no effect), and Cr(VI) had no effect. Chromium(VI) at 1,250 mg/kg was ineffective when worms were introduced after the soil had equilibrated for 2 weeks, regardless of the length of exposure.

Survival may be more sensitive than reproduction to the metal when it is added to the earthworm substrate as a soluble salt. The relative toxicity of Cr(III) and Cr(VI) is not clear from these studies. Cr(VI) ions can pass through cell membranes with much greater ease than Cr(III) ions. However, it is thought that Cr(VI) is reduced to Cr(III) inside the cell (Molnar et al. 1989); this latter may be the final active form. Without a better understanding of Cr transformations in the soil, transport across earthworm cell membranes, and reactions within the cell, it is difficult to separate the effects of the two different forms. There is not enough information for the development of a soil invertebrate Eco-SSL for chromium (EPA, March 2005).

It is generally agreed that suspended particulates are a major source of transport in aquatic systems. Chromium toxicity to aquatic biota is significantly influenced by abiotic

variables such as hardness, temperature, pH, and salinity of water. Aquatic organisms were found to be more sensitive to chromium VI than chromium III. LC_{50} values ranged from 445 ppb to 3,100 ppb for freshwater crustaceans, rotifers, and marine crustaceans (USDI, 1986).

Fish

It is generally agreed that suspended particulates are a major source of transport in aquatic systems. Chromium toxicity to aquatic biota is significantly influenced by abiotic variables such as hardness, temperature, pH, and salinity of water. There is no indication of biomagnification of chromium along the aquatic food chain (ATSDR, 1991). Sensitive species of freshwater aquatic organisms showed reduced growth, inhibited reproduction, and increased bioaccumulation at 10.0 µg/L of Cr(VI) and other adverse effects at 30.0 ug/L of Cr(III). Cr(III) is more acutely toxic to fish than Cr(VI), while the converse is true for chronic toxicity (i.e., Cr(VI) is more chronically toxic than Cr(III)) (NRC, 1976). Cr(III) is less damaging than Cr(VI) (Eisler, 1986). Rainbow trout are able to regulate Cr in smaller doses, by both active and passive measures. Active measures include reduced absorption and/or increased urination. The passive method is limiting the number of binding sites for Cr *in vivo* (Buhler et al., 1977). Cr(III) is poorly absorbed through the intestinal tract (less than 1% of an oral dose), where Cr(VI) is absorbed from 3-6% (Langard and Norseth, 1979).

Terrestrial Vertebrates

The current Eco-SSLs for protection of birds is 26 mg/kg (Cr III). The Eco SSLs for protection of mammals are and 34 mg/kg (CrIII) and 81 mg/kg (CrVI) (EPA, March 2005).

Mammals

Under certain conditions, chromium is a human and animal carcinogenic agent. The biological effects of chromium depend on chemical form, solubility, and valence. In rabbits, both Cr(III) and Cr (VI), given 1.7 mg/kg body weight daily for 6 weeks, adversely affected blood and serum chemistry, and both produced significant morphological changes in liver (Tandon et al., 1978). Laj (1984) found similar results in rats. According to studies of tissue cultures of ovary cells of a Chinese hamster performed by Uyeki and Nishio (1983), the addition of 52 ppb of Cr(VI) both induced the production of sister chromatid exchanges and inhibited cell proliferation. The addition of 520 ppb of Cr(III) did not measurably affect cell proliferation of chromatid exchanges (Uyeki and Nishio, 1983).

Birds

Dietary levels of 10.0 mg Cr(IIII)/kg adversely affected young black ducks, and 5.1 mg Cr(VI)/kg in food and water of mice was associated with elevated tissue residues. Male domestic chickens fed diets containing up to 100 ppm of Cr(VI) for 32 days showed no adverse effects in survival, growth, or food utilization efficiency (Rosomer et al., 1961) however, teratogenic effects were documented in chicken embryos after eggs had been injected with Cr(VI). Deformities included short and twisted limbs, microphthalmia, exencephaly, everted viscera, growth stunting, and parrot beaks (Ridgeway and Karnofshky, 1952; Gilani and Marano, 1979). Adult black ducks fed diets containing 10 or 0 ppm anionic Cr(III) for 5 months were normal in survival, reproduction, and blood chemistry.

The 10 ppm (2.7 mg/kg-bw/da_2) value is the avian LOAEL for this Tier 2 ERA. However, in ducklings from treated groups that were fed Cr-contaminated diets at original parental dosages, growth patterns were altered and survival was reduced (Haseltime et al., 1985).

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COPPER

Environmental Fate and Transport

Sources

Copper is an important metal that occurs naturally in rock, soil, water, sediment, air, and in plants and animals where it is an essential element. In its pure form, copper is used as the primary metal or an alloy in the manufacture of wire, sheet metal, pipes, etc. Copper is also combined with other elements to form copper compounds. Copper sulfate, the most abundant copper compound, is used as a fungicide on citrus, peanuts, potatoes, and other vegetable crops. Copper sulfate and other copper compounds are also used as algicides, for electroplating, in petroleum refining, and in the production of copper arsenate wood preservatives, azo dye, and the manufacture of other copper compounds (Agency for Toxic Substances and Disease Registry [ATSDR], 1989).

In its pure form, copper exists as a reddish solid with a molecular weight of 63.546; it has a melting point of 1083.4^{o}C and a boiling point of 2567^{o}C. It is insoluble in water and has a specific gravity of 8.92. Copper sulfate, a blue-white solid, has a molecular weight of 159.60, a specific gravity of 3.603, and begins to decompose at temperatures above 200^{o}C. Copper sulfate is relatively insoluble in water but soluble in ethanol and methanol (Weast, 1980).

Fate and Transport

The amount of copper released to the air is only a small fraction of the total copper release to the environment (Perwack et al., 1980). The primary source of atmospheric copper is windblown dust; however, other natural sources such as volcanoes, decaying vegetation, forest fires, and sea spray contribute (Davies and Bennet, 1985). Human uses of copper in the production of wood, oil and gasoline consumption, and the manufacture of phosphate fertilizer are also sources of copper emissions.

Atmospheric copper is in the form of particulate matter or is adsorbed to particulate matter. Particle size is a major determinant of its fate, which may be deposition on the earth's surfaces by precipitation and gravitational settling (Chan et al., 1986). Copper binds tightly to soils. Leaching may occur in sites where acid rain is significant (Strain et al., 1984); however, in most cases, copper remains in the top layers of the soil. Of course the physical and organic characteristics of the soil, as well as the form of copper present, affect its mobility (ATSDR, 1989).

Speciation and Bioavailability

Unstable forms of copper present in water tend to complex with organic and inorganic ligands like ammonia and chloride ions, and humic substances. Insoluble, stable forms of copper bind particulate matter and settle onto aquatic sediments. Copper contamination in drinking water is generally derived from copper pipes and brass faucets that have held tap water overnight (ATSDR, 1989).

In all studies examined, copper was not found to bioaccumulate or bioconcentrate in the food chain. Bioconcentration factor estimates for fish yielded low potentials; however, bottom dwelling aquatic species such as oysters have quite high potentials for bio-concentration. Regardless, there is no evidence of bioaccumulation, even in bottom dwelling species (Perwack et al., 1980).

Bioaccumulation was examined in several trophic levels of terrestrial vertebrates, and no evidence of bioconcentration in the food chain was found (Hernandez et al., 1985). Likewise, examination of terrestrial invertebrates at the lower trophic levels indicated that organismal concentration of copper was poorly correlated with soil concentration (Beyer and Cromartie, 1987).

Toxicological Profile

Plants

The most common toxicity symptoms in terrestrial plants include reduced growth, poorly developed root systems, and leaf chlorosis. Copper interferes with enzyme functioning in the roots and interferes with photosynthesis and fatty acid synthesis (Sample et al., 1997). In tests of plants in sandy soil, root and shoot weight and growth were significantly impacted at concentrations of 100 mg/kg as CuSO4 (Miles and Parker, 1979). In a loam soil leaf weight of bush beans were reduced by 26% at a soil concentration of 200 mg/kg, while 100 mg/kg had no effect (Wallace et al., 1977).

Growth and plant weight of red pine, maple, dogwood, and cedar were affected from chronic exposures to 4 mg/L Cu from CuSO4 in nutrient solution (Heale and Ormrod, 1982). Other studies demonstrated reduced growth in various plants at concentrations of copper as low as 0.031 mg/L in nutrient solution (Wong and Bradshaw, 1982). Will and Suter (1995) reported no observable effect concentration (NOEC) and lowest observable effect concentration (LOEC values for the toxicity of copper to plants in solution, with NOECs from 0.5 to 50 mg/L and LOECs from 0.031 to 100 mg/L. The current Eco-Soil Screening Level (SSL) for the protection of plants is 70 mg/kg (United States Environmental Protection Agency [EPA], July 2006).

Copper salts are commonly used to control algae with toxicity values for aquatic plants ranging from 0.001 to 10.45 mg/L for 32 tests (EPA, 1985). Copper sulfate and other copper compounds are effective algaecides (free copper ions are the lethal agent). Single-cell and filamentous algae and cyanobacteria are particularly susceptible to the acute effects, which include reductions in photosynthesis and growth, loss of photosynthetic pigments, disruption of potassium regulation, and mortality. Sensitive algae may be affected by free copper at low (parts per billion) ppb concentrations in freshwater (ATSDR, 1990). The lowest chronic screening value for freshwater aquatic plants was 1 µg/L (Suter and Tsao, 1996).

Invertebrates

Various studies of copper toxicity to earthworms determined that concentrations below 100 mg/kg do not typically demonstrate toxicity, while concentrations >500 or 1000 mg/kg have

demonstrated significant toxicity (Sample et al, 1997). The current Eco-SSL for the protection of soil invertebrates is 80 mg/kg (EPA, July 2006).

Copper is highly toxic in aquatic environments with toxic effects in invertebrates (Horne and Dunson, 1995). The toxicity appears to be a function of calcium hardness and carbonate alkalinity (Sample et al. 1997). Suter and Tsao (1996) report the lowest chronic screening value for daphnids and non-daphnid invertebrates in freshwater as 0.23 μg/L and 6.066 μg/L, respectively.

Fish and Amphibians

Copper is highly toxic in aquatic environments and has effects in fish, invertebrates, and amphibians, with all three groups equally sensitive to chronic toxicity (Horne and Dunson, 1995). The toxicity appears to be a function of calcium hardness and carbonate alkalinity (Sample et al., 1997). The lowest chronic screening value for freshwater fish from exposure to copper is 3.8 μg/L (Suter and Tsao, 1996). Copper is highly toxic to amphibians (including mortality and sodium loss), with adverse effects in tadpoles and embryos (Horne and Dunson, 1995; Owen, 1981).

Terrestrial Vertebrates

The current Eco- SSLs for the protection of birds and mammals are 28 mg/kg and 51 mg/kg, respectively (EPA, July 2006).

Mammals

Most copper salts occur in two valence states, as cuprous (Cu+) and cupric (Cu2+) ions, with the divalent state as the biologically available and toxic form (ATSDR, 1990). Copper is metabolized and transferred to various organic ligands, with the liver being the primary organ involved in metabolism and storage (ATSDR, 1990). Animal studies showed that oral exposure results in hepatic and renal accumulation of copper with liver and kidney necrosis at 100 mg/kg-day and hematological effects at doses of 40 mg/kg-day (Sample et al. 1997). Copper can increase fetal mortality and developmental abnormalities. Sub-chronic and chronic exposure of mammals to copper demonstrated increased mortality, depressed growth, reduced litter size and decreased fetal weights (Sample et al., 1997).

Mammals are not as sensitive to copper toxicity as aquatic organisms; however, toxicity in mammals includes a wide range of animals and effects such as liver cirrhosis, necrosis in kidneys and the brain, gastrointestinal distress, lesions, low blood pressure, and fetal mortality (ATSDR, 1990; Kabata-Pendias and Pendias, 1992; Ware, 1983; Vymazal, 1995).

Birds

Toxic effects in birds include reduced growth rates, lowered egg production, and developmental abnormalities (ATSDR, 1990). Birds tend to tolerate copper exposure better than mammals, with few studies noting adverse effects until approximately 500 mg/kg in the diet (Sample et al., 1997). Mallards exposed to >55 mg/kg-day $CuCO_3$ demonstrated toxic effects while doses of <29 mg/kg-day showed no symptoms (Pullar, 1940).

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4,4'-DDD, 4,4'-DDE and 4,4'-DDT

Environmental Fate and Transport

Sources

4,4'-DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane) was a widely used chemical to control insects on agricultural crops and insects that carry diseases like malaria and typhus. 4,4'-DDD (1,1-dichloro-2,2-bis(p-chlorophenyl)ethane) was also used to control pests, but to a far lesser extent than 4,4'-DDT. One form of 4,4'-DDD has been used medically to treat cancer of the adrenal gland. Both 4,4'-DDE and 4,4'-DDD are breakdown products of 4,4'-DDT.

4,4'-DDT does not occur naturally in the environment. After 1972, the use of 4,4'-DDT was no longer permitted in the United States except in cases of a public health emergency. The use of 4,4'-DDD to kill pests has also been banned.

Fate and Transport

Large amounts of 4,4'-DDT were released into the air and on soil or water when it was sprayed on crops and forests to control insects. 4,4'-DDT may still be released into the atmosphere in other countries where is still manufactured and used, including Mexico. 4,4'-DDT, 4,4'-DDE, and 4,4'-DDD may also enter the air when they evaporate from contaminated water and soil.

4,4'-DDT, 4,4'-DDE, and 4,4'-DDD last in the soil for a very long time. Eventually, most of 4,4'-DDT breaks down into 4,4'-DDE and 4,4'-DDD, generally by the action of microorganisms. 4,4'-DDE and 4,4'-DDD also last in the soil for long periods. These chemicals may also evaporate into the air and be deposited in other places. They stick strongly to soil, therefore generally remain in the surface layers of soil. Some soil particles with attached 4,4'-DDT, 4,4'-DDE, or 4,4'-DDD may get into rivers and lakes in runoff. Only a very small amount, if any, will seep into the ground and get into ground water. The length of time that 4,4'-DDT will last in soil depends on many factors including temperature, type of soil, of time that 4,4'-DDT will last in soil depends on many factors including temperature, type of soil, and whether the soil is wet. 4,4'-DDT lasts for a much shorter time in the tropics where the chemical evaporates faster and where microorganisms degrade it faster. 4,4'-DDT appears faster when the soil is flooded or wet than when it is dry. 4,4'-DDT disappears faster when it initially enters the soil. Later on, evaporation slows down and some 4,4'-DDT moves into spaces in the soil that are so small that microorganisms cannot reach the 4,4'-DDT to break it down efficiently. In tropical areas, Σ4,4'-DDT may disappear in much less than a year. In temperate areas, half of the Σ4,4'-DDT initially present usually disappears in about 5 years. However, in some cases, half of the Σ4,4'-DDT initially present will remain for 20, 30, or more years.

4,4'-DDT and its metabolites may be transported from one medium to another by the processes of solubilization, adsorption, remobilization, bioaccumulation, and

volatilization. In addition, 4,4'-DDT can be transported within a medium by currents, wind, and diffusion. 4,4'-DDT and its metabolites are only slightly soluble in water. Therefore, loss of these compounds in runoff is primarily due to transport of particulate matter which these compounds are bound. The amount of 4,4'-DDT transported into streams as runoff is dependent on the methods of irrigation used. Since the compounds are bound strongly to soil, 4,4'-DDT would remain in the surface layers of soil and not leach into groundwater. However 4,4'-DDT, can adsorb to free-moving dissolved organic carbon, a soluble humic material that may occur in the soil solution. This material behaves as a carrier and facilitates transport of 4,4'-DDT into subsurface soil. 4,4'-DDT released into water adsorbs to particulate matter in the water column and sediment.

Speciation and Bioavailability

Under simulated atmospheric conditions, both 4,4'-DDT and 4,4'-DDE decompose to form carbon dioxide and hydrochloric acid (World Health Organization [WHO], 1979). In air and sunlight, 4,4'-DDT is subject to direct photooxidation and reaction with photochemically produced hydroxyl radicals. Biodegradation may occur under both aerobic and anaerobic conditions in the presence of certain soil microorganisms; including fungi, algae, and mixed microbial populations (Lichtenstein and Schulz, 1959; Stewart and Chisholm, 1971; Verma and Pillai, 1991). Although it is known that exposure to these compounds can incur in the environment, there is a lack of data to quantify the bioavailability. In particular, it is known that fish and some plants bioaccumulate these compounds and that those who consume these fish and plants will incur some exposure to these compounds. However, because of universal body burdens of these compounds, the contribution of any particular media, especially soil and sediment, is not clearly understood.

It is not clear why the 4,4'-DDT levels are higher in organisms living at greater depths since 4,4'-DDT appears to be evenly distributed in the water column. Since 4,4'-DDT adsorbs to o particulate matter that sinks into the sediment, as with detritus from aquatic organisms, fish, and other organisms living at the bottom of the sea may accumulate higher levels of 4,4'-DDT than organisms living at the surface because their food chain is associated with benthic feeders. Regional differences in 4,4'-DDT levels in biota may be associated with the productivity of the ocean and greater sedimentation of detritus from aquatic organisms. Arctic mammals feeding on 4,4'-DDT-contaminated bioaccumulate the chemical in their fat.

Toxicological Profile

Plants

Plants can take up 4,4'-DDT from soil and air and store it in their leaves and roots. 4,4'-DDT as a pesticide used on crops is not toxic to plants. However, phytotoxicity of 4,4'-DDT to cucurbits, beans, young tomatoes, and some varieties of barley is reported (Hartley and Kidd, 1987).

Aquatic Invertebrates

The available data for 4,4'-DDE indicate that the acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,050 μg/L.

Fish

4,4'-DDT is highly toxic to fish. However, widespread bird kills have resulted from bioconcentration of 4,4'-DDT through food chains, i.e., from fish or earthworms. The mortality of cased and free-living young salmon in the streams of forests sprayed with 4,4'-DDT was studied. A single application of 4,4'-DDT at 0.5 lb/acre caused heavy loss of under yearling salmon and parr within 3 months. At 0.25 lb/acre, there was no apparent effect on parr, but all the under yearlings were killed. Further, 2 applications at 0.25 lb/acre each at 10-day intervals were as harmful as a single application of 0.5 lb/acre. Besides, 4,4'-DDD at 0.25 or 0.5 lb/acre or malathion at 0.125 lb/acre was as harmful as 4,4'-DDT at 0.25 lb/acre (Murty, 1986).

Amphibians

Lethality information in adult amphibians is limited to studies in the common frog and bullfrog. No mortality was seen in common frogs dosed twice weekly for 8 weeks with 4,4'-DDT at 0.6 mg/kg, but in treated frogs that were not fed, 50% mortality was seen by the end of the exposure period. The LD_{50} in the adult common frog 20 days after a single oral administration of 4,4'-DDT in gelatin capsules at unreported dose levels was estimated to be 7.6 mg/kg body weight; LD_{50} values at 3 and 4 days after the single oral administration were approximately 85 and 25 mg/kg respectively. Mortality was seen in common frog tadpoles immersed for 1 hour in 1 or 10ppm 4,4'-DDT, but not in $\leq$ 0.1 ppm. In the adult bullfrogs, 14-day oral LD_{50} of >2,000 mg/kg body weight was reported following a single oral administration of 4,4'-DDT in gelatin capsules at unreported dose levels.

Terrestrial Vertebrates

In animals, 4,4'-DDT produces embryotoxicity and fetotoxicity, but not teratogenicity. In several studies, lethal dose of these compound on mammals have been reported. Historically, observations of high mortality rate in local wild bird populations occurred coincidentally with application of 4,4'-DDT for pest control.

Mammals

In bats, no mortality occurred after single oral doses of 4,4'-DDT below 45 mg/kg weight for up to 31 days postdosing, but 100% mortality occurred within 28 days in groups administered single doses of $\geq$ 95 mg/kg body weight; the LD50 was 63 mg/kg body weight. A single oral dose of technical grade 4,4'-DDT at 20 mg/kg body weight caused some mortality in big brown bats, while > 40 mg/kg body weight was 100% lethal. Clark (1981) estimated that the minimum lethal concentrations are 12 ppm (w/w) 4,4'-DDT in the brain of the little brown bat, and 460 and 540 ppm 4,4'-DDE in the free-tailed bat and the little brown bat, respectively.

A single oral dose of >237 mg technical grade 4,4'-DDT/kg caused death to mice (Kashyap et al., 1977). The LD_{50} values reported in rats exposed to single oral doses of 4,4'-DDT ranged from 113 to 800 mg/kg (Ben-Dyke *et al.,* 1970). The LD_{50} values for guinea pigs and rabbits after oral exposure to 4,4'-DDT were 400 mg/kg and 300 mg/kg, respectively (Cameron and Burgess, 1945). The nervous system appears to be one of the primary targets in animals after acute subchronic, and chronic oral exposure to 4,4'-DDT (Herr *et al.,* 1985; Pranzatelli and Tkach, 1992). Intermediate exposures, in which animals were exposed to 4,4'-DDT in food, caused cancer increases in mice but not in rats or hamsters. Green (1969) fed male and female rats diets providing an intake of 0.35 mg 4,4'-DDT/kg/day for 60 days before mating and found a 75% depression of fertility but no effect on litter size. A decrease in fertility was observed in female rats fed technical grade 4,4'-DDT for 60 days at dose levels of 26 mg/kg/day (Bernard and Gaertner, 1964). Intermediate oral exposure to 4,4'-DDT in animals has been shown to produce developmental effects such as infertility, mortality, and slow development in offspring of exposed dams (Clement and Okey, 1974).

Death occurred in mice after single oral doses of o,p'-4,4'-DDE ranging from 810 to 880 mg 4,4'-DDE/kg (Tomatis *et al.,* 1974). There are several studies of the potential carcinogenicity of 4,4'-DDE and 4,4'-DDD in rats, mice, and hamsters. 4,4'-DDE administered chronically in the diet produced liver tumors in mice at doses of 19-34 mg/kg/day for 30-78 weeks (Tomatis *et al.,* 1974) and in hamsters dosed at 40 mg/kg/day for 124 weeks (Rossi et al., 1983). An LD_{50} was reported for rats as a range of single oral doses (400-4,000 mg/kg) in which mortality was observed in 50% of rats exposed to 4,4'-DDD (Ben-Dyke *et al.,* 1970). An LD_{50} for 4,4'-DDD in rats of > 4,000 mg/kg was reported by Gaines (1969). Tomatis *et al.* (1974) reported an LD_{50} in mice after a single oral dose ranging from 1,466 to 1,507 mg 4,4'-DDD/kg.

For 40 days, free-tailed bats were fed mealworms that were raised in wheat bran containing 100 ppm 4,4'-DDE; the treated bats lost body weight quicker and died sooner than an untreated control group during postexposure starvation period. Among the 17 treated bats, a strong negative relationship was seen between 4,4'-DDE residue in the brain and percent lipid in the carcass, suggesting that 4,4'-DDE mobilized from fat will accumulate in the brain. For 40 days, free-tailed bats were fed mealworms that were raised in wheat bran containing 100 ppm 4,4'-DDE; the treated bats lost body weight quicker and died sooner than an untreated control group during postexposure starvation period.

Birds

Historically, observations of high mortality rate in local wild bird populations occurred coincidentally with application of 4,4'-DDT for pest control. Several authors have postulated that high mortality may occur during times of stress, such as during nesting or during migration, when energy from fat stores is metabolized. As fat stores are depleted and newly absorbed 4,4'-DDT could distribute to the brain; as in mammals, accumulation of high levels in the brain of birds is hypothesized to be lethal. Since 4,4'-DDT was banned, the primary route of exposure to 4,4'-DDT compounds in wild bird populations has been in the diet through the food chain. Available experimental data on bird lethality

indicate that 4,4'-DDT/4,4'-DDE/4,4'-DDD have moderate to low toxicity in birds after ingestion in the diet or from gavage administration.

Acute LD_{50} values of orally administered 4,4'-DDT in 2-month-old Japanese quail, 4,4'-DDT in 4-month-old pheasant technical grade 4,4'-DDT in 6-month-old California quail, 4,4'-DDT in 3-month-old Mallard ducks, 4,4'-DDT in rock dove, and 4,4'-DDT in adult sandhill crane ranged from 595 mg/kg body weight in 6-month-old male California quail to >4,000 mg/kg body weight in male and female rock doves. Dietary LC_{50} values for 4,4'-DDT ingestion ranged from 311 to 1,869 mg/kg diet after 5-day exposures in immature bobwhite quail, Japanese quail, Mallard duck, and pheasant. As early as 1950 it was found that large subcutaneous doses of 4,4'-DDT in young roosters (300 mg/kg/day) inhibited testicular growth and development of secondary sexual characteristics (Hayes, 1982) and intermediate exposure to 4,4'-DDT affected fertility.

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ENDRIN/ENDRIN KETONE

Environmental Fate and Transport

Sources

Endrin is a solid, white, almost odorless substance that was used as a pesticide to control insects, rodents and birds. Endrin was introduced in the United States in 1951 as an avicide, rodenticide, and insecticide. Its principal use to control the cotton bollworm and tobacco budworm peaked in early 1970s. Endrin has not been produced or sold for general use in the United States since 1986. Endrin ketone is a breakdown product of endrin, when it is exposed to light.

Transport and Fate

No studies on the environmental fate of endrin aldehyde or endrin ketone could be found in the available literature. Endrin ketone may react with photochemically generated hydroxyl radicals in the atmosphere, with an estimated half-life of 1.5 days. Available estimated physical/chemical properties of endrin ketone indicate this compound will not volatilize from water; however, significant bioconcentration in aquatic organisms may occur. In soils and sediments, endrin ketone is predicted to be virtually immobile; however, detection of endrin ketone in groundwater and leachate samples at some hazardous waste sites suggests limited mobility of endrin ketone in certain soils.

Endrin does not dissolve well in water. It has been found in ground water and surface water, but only at very low levels. It is more likely to cling to the bottom sediments of rives, lakes, and other bodies of water. Endrin is generally not found in the air except when applied to fields during agricultural applications.

The persistence of endrin in the environment depends highly on local conditions. Some estimates indicate that endrin can stay in soil for over 10 years. Endrin may also be broken down by exposure to high temperatures (230 ^{0}C) or light to form primarily endrin ketone and endrin aldehyde. Endrin tends to persist in the environment mainly in forms sorbed to sediments and soil particles. A conservative estimate of its half-disappearance time in sandy loam soils is approximately 14 years (41% of endrin applied still remained in the soil after 14 years).

Migration of endrin into groundwater would not generally be expected from normal agricultural application. However, endrin has been detected in some groundwater, suggesting that leaching may be possible in some soils under certain conditions. Biodegradation does not appear to be a significant fate process for endrin in soils. Hydrolysis in moist soils is also not expected to be significant. In combination, losses from volatilization, photodegredation, and heat transformation account for the rapid decrease in endrin residues in soil surfaces exposed to bright sunlight.

In spite of its low vapor pressure, endrin has been found to volatilize significantly (20-30%) from soils within days after application. In air, endrin will be primarily absorbed to particulates, which may be re-entrained to soil or surface water via wet or dry deposition.

Laboratory studies have indicated that a predominant mechanism for the transformation and degradation of endrin in air under field conditions is via photochemical reactions and rearrangements to yield primarily endrin ketone, with minor amounts of endrin aldehyde. Endrin may also be transformed by heat in the atmosphere, yielding primarily the pentacylic ketone and endrin aldehyde. Endrin may also react with photochemically generated hydroxyl radicals in air, with a predicted half-life ranging from 1.45 hours to 1.8 days.

Endrin may be transported from soil to surface water via runoff or irrigation. When released to water, endrin strongly adsorbs to sediment and bioconcentrates significantly in aquatic organisms.

Endrin may be biodegraded in water, but most laboratory studies indicate that this will not be a significant fate process. In addition, neither hydrolysis nor volatilization is a significant fate process for endrin in water. The half-life of endrin in water is more than 4 years. Degradation of endrin in soils under field conditions is not a significant fate process with half-disappearance time of the order of 14 years.

Speciation and Bioavailability

Endrin appears to be biomagnified only slightly through various levels of the food chain. It is likely that endrin released to surface water will undergo photoisomerization to endrin ketone, with minor amounts of endrin aldehyde also being formed.

Isodrin's chemical formula is $C_{12}H_8Cl_6$, which is very similar to endrin's chemical formula, $C_{12}H_8Cl_6O$. The only difference between Isodrin and Endrin is that two of the carbon atoms are attached to oxygen in Endrin. As such Endrin is used as a surrogate for Isodrin.

Toxicological Profile

Summary

Endrin may be transported from soil to surface water via runoff or irrigation. When released to water, endrin strongly sorbs to sediment and bioconcentrates significantly in aquatic organisms. However, endrin appears to be biomagnified only slightly through various levels of the food chain.

Plants

Although no toxicity information was located for plants, uptake of endrin by plants was noted from soils treated as long as 16 years after planting (Nash and Harris, 1973).

Invertebrates

There were minimal invertebrate toxicity studies for endrin. The LC_{50} for the sow bug (*Asellus brevicaudus*), a soil invertebrate, is 1.5 µg/L/95 hr (Johnson et al., 1980). The 96-hr LC_{50} is 3.2 µg/L for the crayfish *Orconectes nais*, 4.2 µg/L for the water flea *Daphnia magna*, and 20 µg/L for the water flea *Daphnia pulex* (Johnson et al., 1980).

Fish

Endrin is more toxic to fish than aquatic invertebrates. Endrin toxicity is not appreciably affected by pH, water hardness, or temperature. Several physiological and biochemical variables were altered by endrin, including growth and reproductive development, adrenal and thyroid function, serum electrolyte balance and osmoregulation, glycogen metabolism, serum protein composition, resistance to stress, and behavioral patterns (Johnson et al., 1980).

The 96-hr LC_{50} is 0.31 µg/L for largemouth bass, 0.32 µg/L for common carp and channel catfish, 0.61µg/L for bluegill, 1.1 µg/L for mosquitofish, and 1.8 µg/L for fathead minnow (Johnson et al., 1980). In fish, endrin residues accumulate rapidly by dietary or bath exposure, reaching levels of 400 to 2000 times the exposure level.

Terrestrial Vertebrates

Mammals

Ingestion of endrin can cause central nervous system effects (hyperexcitability, convulsions), abnormal bone formation in fetuses, and nonspecific degeneration of the liver, kidney and brain, and death (Agency for Toxic Substances and Disease Registry [ATSDR], 1996)

Three known mammalian metabolites of endrin are more toxic than the parent compound. The LD_{50} of 12-ketoendrin is 1.1 and 0.8 mg/kg in male and female rats respectively, and it exerts its full effect during the first 20 hr, compared to 4 to 8 days for endrin. Thus, 12-ketoendrin may be responsible for much of the acute toxicity of endrin or of intermediate metabolites in the rat. However, the fact that the brains of rats killed by endrin contain (in addition to endrin) substantially less 12-ketoendrin than do the brains of rats killed by 12-ketoendrin suggests that endrin is toxic per se (Hayes, 1982).

Oral LD_{50} ranged from 6 to 50 mg/kg in mammals (Hudson et al., 1984, Treon et al., 1955). The lowest chronic lowest observed adverse effect level (LOAEL) for reproduction (reduced litter size, fetal mortality) was 0.65 mg/kg; significant increase in mortality among the parent males and females also was seen (Good and Ware, 1969).

Birds

Signs of endrin intoxication in birds included ataxia, slowness, drowsiness, tremors, trachael congestion, prostration, convulsions, wing-beat convulsions, and opisthotonos (Hudson et al., 1984). Oral LC_{50} values (ppm active ingredient in diet) were 14 ppm for ring necked pheasant and bobwhite quail, and 18 ppm for Japanese quail and mallard (Hill et al., 1975). Oral LD_{50} values were 1.06 mg/kg for sharp-tailed grouse, 1.19 mg/kg for California quail, 1.78 mg/kg for pheasant, 2 – 5 mg/kg for rock dove, and 5.64 mg/kg for mallard (Hudson et al. 1984). The lowest chronic LOAEL for reproduction (reduced egg production and hatching success) was 0.1 mg/kg for the screech owl (Fleming et al., 1982).

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HEXACHLOROBENZENE

Environmental Fate and Transport

Sources

Hexachlorobenzene is a white crystalline solid that is not very soluble in water. It does not occur naturally in the environment. It is formed as a by-product while making other chemicals, in the waste streams of chloralkali and wood-preserving plants, and when burning municipal waste. Hexachlorobenzene was widely used as a pesticide to protect the seeds of onions and sorghum, wheat, and other grains against fungus until 1965. It was also used to make fireworks, ammunition, and synthetic rubber (ATSDR, 2002).

Fate and Transport

Hexachlorobenzene is among the most persistent environmental pollutants because of its chemical stability and resistance to degradation. If released to the atmosphere, hexachlorobenzene exists primarily in the vapor phase and degradation is extremely slow. If released to water, hexachlorobenzene will partition from the water column into sediment and suspended particulate matter. The half-life value of hexachlorobenzene is estimated to range from 3 to 6 years in surface water and from 5 to 11 years in groundwater. If released to soil, hexachlorobenzene will strongly adsorb to organic matter and is generally considered immobile with respect to leaching. Its half life value in soils is estimated to range from 3 to 6 years. Hexachlorbenzene bioaccumlates significantly in both terrestrial and aquatic food chains. The bioaccumulative tendencies of hexachlorobenzene have made it a candidate for monitoring in the U.S. Fish and Wildlife Service National Pesticide Monitoring Program and the National Study of Chemical Residues in Fish which was started in 1986. In terrestrial ecosystems, several agricultural crops have been found to accumulate hexachlorobenzene in their roots and in portions growing closest to soil level (ATSDR, 2002).

Toxicological Profile

Summary

Hexachlorobenzene is slightly to moderately toxic to bird species. In Japanese quail it has a 5-day dietary LC50 of 568 ppm (Hill, 1986). The reported acute oral LD50 values in bobwhite quail were 575 mg/kg and in mallard duck was 1450 mg/kg (HSDB,1995). Hexachlorobenzene is slightly toxic to fish species, with reported 96-hour LD50 values of 11 to 16 mg/L in channel catfish, greater than 50 mg/L in coho salmon, 22 mg/L in fathead minnow, and 12 mg/L in bluegill and large mouth bass (Johnson, 1980).

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LEAD

Environmental Fate and Transport

Sources

Global production of lead from both smelter and mining operations has been high throughout this century. Lead is commonly used in storage batteries as well as ammunition, solder, and casting materials. In addition, tetraethyl lead was a principal additive to gasoline as an anti-knock agent, and was commonly used as an additive in paints. In short, lead is one of the most ubiquitous pollutants in the civilized world. Release of lead to air is no less than the releases of lead to soil.

Fate and Transport

Uptake of lead from soil by plants is not significant. In animals, most dietary lead is passed through the body, but the small amounts absorbed can accumulate and be harmful. However, levels of lead can build up in plants and animals exposed to lead contaminated air, water, or soil for long periods of time.

Lead is strongly sorbed in sediments and the rate is strongly correlated with grain size and organic content. In the absence of soluble complexing species, lead is almost totally adsorbed to clay particles at pHs greater than 6 (Moore and Ramamoorthy, 1984). Biomethylation of lead in sediments makes some of the lead bioavailable to benthic invertebrates (Irwin et al. 1997).

Speciation and Bioavailability

In surface water, lead is most soluble and bioavailable under conditions of low pH, low organic content, low levels of suspended solids, and low levels of salts of calcium, iron, manganese, zinc, and cadmium. In surface water, lead exists in three forms, dissolved labile (e.g., Pb^{+2}, $PbOH^{+}$, $PbCO_3$), dissolved bound (e.g., colloids or strong complexes), or as a particulate (Benes et al. 1985). Most lead in natural waters is precipitated to the sediment as carbonates or hydroxides. Lead in sediment is mobilized and released when the pH decreases suddenly or ionic composition changes (Demayo et al. 1982). Some Pb^{+2} in sediments may be transformed to tetraalkyllead compounds, including tetramethyllead, through chemical and microbial processes. Methylation of lead in sediment is positively related to increasing temperatures, reduced pH, and microbial activity, and is independent of lead concentration (Demayo et al. 1982). The concentration of tetraalkylleads in sedments is low, representing less than 10% of total lead.

There is some evidence of bioaccumulation through the food web of organic forms of lead, such as tetraethyl lead. The majority of benthic invertebrates do not bioconcentrate lead from water or abiotic sediment particles. Lead tends to bioconcentrate in mussels and clams, but not in fish (Irwin et al. 1997). Bioavailability of lead in soils to plants is limited, but is enhances by reduced soil pH, reduced content of organic matter and inorganic colloids, reduced iron oxide and phosphorus content, and increased amount of lead in soil. Lead is taken up by plants through active transport through the roots and by absorption of

lead adhered to foliage (Boggess 1997). Lead concentration is always higher in the roots and stems than in the younger shoots or flowers. There is no evidence that plants are important in food chain biomagnification of lead (EPA 1980).

Toxicological Profile

Plants

Generally, damage to plants is negligible. However, at soil lead levels above 200 mg/kg, lead may reduce crop yields. There are lead-resistant and lead-sensitive breeds of any plant species. In some species, there is a threshold soil concentration before there will be elevated concentrations in the plant (Eisler April 1988). In emergent plants, lead inhibits growth, reduces photosynthesis, and reduces mitosis and water absorption (Demayo et al. 1982). In two weed species (*Cassia spp.*), pollen germination was reduced by 90% and seed germination by 87% at lead soil concentrations of 500 mg/kg dry weight and plant concentration of 300 mg/kg dry weight in foliage (Krishnayya and Bedi 1986).

Soil Invertebrates

Concentration of lead was higher in earthworms near highways than away from highways, but toxicity of lead in earthworms was not determined (Beyer and Moore 1980). However, survival and reproduction in woodlice, a littoral invertebrate, was reduced at 12,800 mg lead/kg soil litter, as lead oxide, in an oral toxicity test for 64 weeks, or two generations (Beyer and Anderson 1985).

Aquatic Invertebrates

Water hardness is a critical factor governing the solubility and toxicity of lead. Both the acute and chronic toxicity of lead increase with decreasing water hardness (i.e., lower concentrations of lead are sufficient to elicit toxic responses in soft water than in hard water) as lead becomes more soluble and bioavailable to aquatic organisms. For example, the cladoceran *Daphnia magna* is three times more sensitive to lead in soft water than in hard water (Chapman et al. Manuscript). Biesinger and Christensen (1972) reported a16% impairment in reproduction for *Daphnia magna* at 30 µg/L lead chloride in soft water (hardness 45 mg/L $CaCO_3$) after 21 days (chronic study). The lowest reported LC_{50} was 28.5 µg/L over a 28-day exposure at a hardness of 46 mg/L $CaCO_3$ for the amphipod *Gammarus pseudolimnaeus* (Spehar et al. 1978).

The influence of pH on lead toxicity in freshwater invertebrates is less clear. Cladocerans (*Ceriodaphnia dubia*) and amphipods (*Hyalella azteca*) were more sensitive to lead toxicity at pH 6 to 6.5 than at higher pH (Schubauer-Berigan et al. 1993). Lead was 100 times more toxic to the amphipod, *H. azteca*, at a pH range of 5.0 to 6.0 (Mackie 1989) than at a pH range of 7.0 to 8.5 (Schubauer-Berigan et al. 1993). Mortality increased with decreased pH in the bivalve, *Pisidium casertanum*, while pH-independent mortality was reported for gastropods and crustacean under similar exposure conditions (Mackie 1989).

Amphibians

Eisler (1988) reported that tadpoles of bullfrogs (*Rana catesbeiana*) and green frogs (*Rana clamitans*) from drainage ditches along highways had elevated amounts of lead (up to 270 mg/kg dry weight), which positively correlated with the lead in the sediments and with the average daily traffic volume. Diets with amounts of lead similar to those in tadpoles collected along heavily traveled highways have caused adverse physiological and reproductive effects in some species of birds and mammals (Birdsall et al. 1986).

Lead poisoning in adult leopard frogs (*Rana pipiens*) is indicated by a series of signs: sloughing of integument; sluggishness; decreased muscle tone, decreases in red blood cells, white blood cells, neutrophils and monocytes; erosion of the gastric mucosa; and (before death) excitement, salivation, and muscular twitching. The 30-day LC_{50} value of *R. pipiens* was 105 mg/L, with some deaths and elevated liver residues noted at 25 mg/L (Kaplan et al. 1967). At water concentrations as low as 0.5 mg/L, physiological effects were seen in frogs and salamanders (Eisler 1988).

Terrestrial Vertebrates

Tetraalkyllead mode of action differs from that of inorganic lead. Organoleads concentrate in the liver, and it is there that tetraalyklleads are probably converted to trialkylleads. Trialkylleads are associated with red blood cells. Tetraalkylleads, by virtue of its liposolubility, accumulate in the nonbony tissues, particularly the brain. In birds, trialkylleads and dialkylleads rapidly traverse biological membranes in the eggs and accumulate in the yolk and developing embryo. The mode of action of organoleads is poorly understood, but is known to inhibit cerebral glucose metabolism and amino acid transport (Eisler April 1988). Lead exerts deleterious effects on hematopoiesis through derangement of hemoglobin synthesis, resulting in a shortened life span of circulating red blood cells, resulting in anemia. Lead accumulates in the kidney and liver reducing function (Eisler April 1988).

Effects of lead on the nervous system are both structural and functional, involving the cerebellum, spinal cord, motor and sensory nerves, nerve cells and ganglia. The result is deterioration of intellectual, sensory, neuromuscular, and physiological functions (Nriagu 1978)

Mammals

In laboratory studies, breeding mice exposed to low doses of lead in drinking water (25 ppm) resulted in loss of the strain in two generations with many abnormalities (Schroeder et al., 1971). In rats, 25 mg/L lead in drinking water during mating and gestation caused developmental effects and deaths in offspring (Kimmel et al., 1980). Blood delta - aminolevulinic acid dehydratase (ALAD) activity associated with exposure to lead was reduced in white-footed mice living near a metal smelter (Beyer et al. 1985). Amounts of whole-body lead content and feeding habits of roadside rodents have been correlated with highest body burdens in insectivores such as shrews; intermediate in herbivores, and lowest in granivores (Boggess, 1977; Getz et al. 1977). Three generations of rats were fed lead acetate for 3 generations at 100 mg/kg with no reproductive effects (Azar et al 1973).

Birds

Most of the information on the effects of lead to terrestrial vertebrates is concerned with the poisoning of waterfowl by lead shot. Apparent symptoms include loss of appetite and mobility, avoidance of other birds, lethargy, weakness, emaciation, tremors, dropped wings, green feces, impaired locomotion, loss of balance and depth perception, nervous system damage, inhibition of heme synthesis, damage to kidneys and liver, and death (Eisler 1988; Mudge 1983). Anemia, kidney disease, testicular and liver lesions, and neurological disorders have been associated with high brain lead concentrations in mourning doves (*Zeneida macroura*) (Kendall 1992). Hatchlings of chickens, Japanese quail, mallards and pheasants are relatively more tolerant to moderate lead exposure, including no effect on growth at dietary levels of 500 ppm and no effect on survival at 2,000 ppm (Hoffman, et al. 1985).

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NICKEL

Environmental Fate and Transport

Sources

Nickel is the 24th most abundant element, occurs naturally in the earth's crust, and appears in all soils to some degree. It is primarily found in the form of oxides and sulfides. It may be released to the environment from natural sources such as volcanoes, forest fires, wind-blown soil, and from human usage, such as processes of industries that make nickel alloys, trash incinerators, oil-burning power plants, and coal-burning power plants. Nickel releases from these human-based sources are often in the form of airborne particulate matter.

Fate and Transport

Large particles may be deposited to land and water through gravitational settling, whereas smaller particles will be deposited by precipitation (Schroeder et al., 1987). Under acidic conditions, nickel is more mobile in the soil and can leach into groundwater. Nickel is a natural constituent in soil, with levels varying depending on the local geology and anthropogenic input. A typical range of soil concentrations is 4 to 80 mg/kg. Nickel adheres tightly to soil particles, especially those containing iron or manganese. Nickel may be transported in to streams and waterways from natural weathering and from anthropogenic sources, where it tends to accumulate in sediment (Agency for Toxic Substances and Disease Registry [ATSDR], 1990). Nickel is more tightly adsorbed to the sediment in alkaline pHs. The absorption of nickel is dependent on its physicochemical form, with water-soluble forms being more readily absorbed.

Speciation and Bioavailability

The metabolism of nickel involves conversion to various chemical forms and binding to various ligands (ATSDR, 1990). Some studies have shown that nickel does not appear to accumulate in plants or small animals living on land that was treated by nickel-containing sludge. Studies have shown that nickel does not appear to concentrate in fish (ATSDR, 1990).

Toxicological Profile

Terrestrial Plants

Plants exposed to nickel-contaminated soil may exhibit stunted growth, wilted leaves, chlorosis, discolored roots, discolored tops, twisted stalks, and thickening of leaf tissue (National Academy of Sciences [NAS], 1975; Frank et al., 1982; World Health Organization [WHO], 1991; Barnum and Bhargava, 1997; Donghua and Wusheng, 1997). Some studies have suggested that terrestrial crop plants may be more sensitive than other plants (Eisler, 1998). Some species have shown effects on growth and chlorophyll metabolism following exposure to only 1 mg/L (Outridge and Scheuhammer, 1993), while 44 mg/kg in soil diminished yields of radishes, beets, cabage, celery, lettuce, and alfalfa (Frank et al., 1982; NAS, 1985).

For freshwater plants, Suter and Tsao (1996) reported the lowest chronic screening value as 5 μg/L. No other information on nickel toxicity to aquatic plants was available.

Invertebrates

The toxicity of nickel to the soil community is dependent upon soil chemistry and the presence of other metals (Babiach and Stotzky, 1982). Various studies of fungi and earthworms have demonstrated toxicity from exposure to nickel.

Various aquatic invertebrates have been shown to be sensitive to nickel. Mollusks and crustaceans tend to be more sensitive than other benthic organisms (Environment Canada, 1994). In freshwater, the lowest chronic screening value for daphnids was <5 μg/L and 128.4 μg/L for non-daphnid invertebrates (Suter and Tsao, 1996).

Fish

Nickel in aquatic environments can cause tissue damage, genotoxicity, and reduced growth (Environment Canada 1994). Nickel exposure in fish can cause damage to gill lamellae, causing blood hypoxia and death (Ellgaard et al., 1995). Four day LC50 values ranged from 5.2 mg/L for fathead minnow (Lind et al., 1978) to 350 mg/L for mosquitofish (Kallangoudar and Patil, 1997). Suter and Tsao (1996) reported the lowest chronic screening value for freshwater fish to be <35 μg/L.

Mammals

Nickel is essential to mammals for iron absorption and growth. Nickel is excreted in the urine and feces with relative amounts for each route being dependent on the route of exposure and chemical form. Most nickel enters the body via food and water consumption. The primary target organ is the kidney for oral exposure (ATSDR, 1990). Other target organs include the cardiovascular system, immune system, and the blood.

Studies on rats administered an oral dose of nickel sulfate hexahydrate caused death in the young of three generations (Oak Ridge National laboratory [ORNL], 1996). Subchronic dietary exposure of rats to nickel produced signs of hematological damages (decreased hematocrit and hemoglobin concentration) and a reduction in weight gain (Whanger, 1973; Clary, 1975). Other investigators also reported degenerative changes in the liver, kidney, and testes (Waltschewa et al., 1972). Nickel chloride, in doses of 1.2 - 20 mg/kg, was found to cause increased resorption sites and malformations in fetuses of treated pregnant females (Storeng and Jonsen, 1981). The EPA classified nickel as a toxic substance from the oral route of exposure.

Birds

Little information on toxicity of nickel to birds is available. A chronic dose of 77.4 mg/kg-day caused no adverse effects while a chronic dose of 107 mg/kg-day caused significant mortality and decreased growth in mallard ducklings (Cain and Pafford, 1981). An acute dose of 3716 mg/kg-day (Hill and Camardese, 1986) and a chronic dose of 0.01 mg/kg-day (NAS, 1975) did not demonstrate adverse effects to Coturnix quail from exposure to nickel.

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POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Environmental Fate and Transport

Sources

Polycyclic aromatic hydrocarbons (PAHs) are ubiquitous in nature, detected in sediment, soil, air, surface water, and plant and animal tissues. They are formed as a result of incomplete combustion of organic materials such as wood, coal, and oil and exist in the environment in quantity, from natural sources. Anthropogenic source are from localized industrial activities associated with large releases of PAHs like coke production, petroleum refining, the manufacture of carbon black, coal tar pitch and asphalt, heating and power generation, and emissions from internal combustion engines. It is estimated that approximately 270,000 metric tons of PAHs reach the environment yearly (Eisler May 1987). The composition of the PAH emissions vary according to the source. PAHs that are particle-bound can be transported long distances and are ultimately removed from the atmosphere through rain and dry deposition. PAHs can bind with soil particles or can leach into groundwater.

Transport and Fate

When released to the atmosphere, PAH compounds will associate with particulate materials. Transport in the atmosphere is dependent upon particulate size, weather conditions, and atmospheric physics. Highly reactive PAHs readily photooxidize. Half-life of PAHs in the atmosphere varies depending upon a number of variables (Eisler May 1987). Much of the PAHs released into the atmosphere reaches the soil by direct deposition or deposition on vegetation. Plants can adsorb or assimilate PAHs and metabolize and degrade the PAHs. However, if the rate of assimilation exceeds metabolism, PAHs can accumulate in plants (Edwards 1983).

Heavier PAHs in the water column will become incorporated into the sediment. Fate of PAHs in sediment is believed to be biotransformation and biodegradation by benthic organisms (EPA 1980). Some PAHs are very persistent in sediment (Neff 1979).

PAHs can be taken into the mammalian body by inhalation, skin contact, or ingestion, although they are poorly absorbed from the gastrointestinal tract. Elimination of PAHs and their metabolites is primarily through the hepatobiliary system and the gastrointestinal tract (Sims and Overcash 1983). In vertebrates, including fish, there is an enzyme (known by various names like mixed-function oxidases or P450-dependent monooxygenases) system that metabolizes PAHs, limiting bioaccumulation up the food chain (West et al. 1984).

Speciation and Bioavailability

PAHs are a diverse group of organic chemicals consisting of substituted and unsubstituted polycyclic and heterocyclic aromatic rings in which interlinked rings have at least two carbon atoms in common (Zander 1983). This results in a wide range of physical and chemical properties, like molecular weight and solubility in water and lipids, which results in a wide range of toxicity, biological effects, bioavailability, and distribution and

persistence in the environment. Lower molecular weight unsubstituted PAH compounds, containing 2 to 3 rings, like naphthalene, fluorenes, phenathracenes, and anthrecenes, have significant acute toxicity to some organisms, while the higher molecular weight 4- to 7-ring aromatics do not (Eisler May 1987).

PAHs are accumulated in terrestrial and aquatic plants and invertebrates, but vertebrates are able to metabolize and eliminate these compounds from their systems. Aquatic animals do not appear to be greatly exposed to PAHs through food chain uptake. Bioavailability of PAHs to plants is decreased with increasing organic soil content (Greenberg 2003).

Fluoranthene and to a lesser extent phenanthrene are found to accumulate in earthworms, a major food source for some terrestrial birds and mammals, exposed to contaminated soil (Ma et al. 1995). Bioaccumulation factors (BAFs) for earthworms exposed to 100 mg/kg phenanthrene range from 0.027 to 0.623. Ma et al. (1995) provides BAFs in earthworms for eleven PAHs from experimental studies. The highest, 18, is for dibenzo(ah)anthracene, followed by 9 for benzo(ghi)perylene. However, for eights of the PAHs, van Brummelen et al. (1996) reports BAFs from field investigations one to two magnitudes lower than Ma et al. (1995).

Toxicological Profile

Summary

Plants

Plants are known to assimilate and uptake PAHs and metabolize them. PAHs are distributed in both the roots and above ground parts (Eisler, May 1987). For terrestrial plants in soil, phytoaccumulation is primarily in the roots with little translocation to other plant parts (Greenberg, 2003).

PAHs at high soil concentrations can be phototoxic to plants. However, organic content of soil decreases plant toxicity from PAHs. PAHs are an example of an environmental toxicant where one environmental factor (light) can enhance risk, while another (binding with organic carbon) can lower risk (Greenberg, 2003). Toxicity tests with terrestrial plants are often difficult to compare because of the variety of test conditions, such as soil vs. hydroponics, soil types, light conditions, and mineral content of soil or water. End points used have been germination, which lacks sensitivity, growth, which is more sensitive but more time consuming, and reproduction, yield, and life-cycle assays, which are cumbersome and very time-consuming (Greenberg, 2003).

Sverdrup, et al. (2003) conducted tests with eight PAHs on seed emergence and early life-stage growth of three terrestrial plants, red clover (*Trifolium pretense*), ryegrass (*Lolium perenne*), and mustard (*Sinapsis alba*). Organic carbon content was 1.6% in a Danish agricultural soil. The PAHs were fluoranthene, pyrene, phenanthrene and fluorene, and N-, S-, and O-substituted analogues of fluorene: carbazole, dibenzothiophene, and dibenzofuran, respectively. Seedling growth was reported to be a far more sensitive end point. Concentrations estimated to give a 20% reduction (EC_{20}) in

seedling freshwater ranged from 36 mg/kg to 290 mg/kg for carbazole, 43 mg/kg to 93 mg/kg for dibenzofuran, 37 mg/kg to 100 mg/kg for dibenzothiophene, 140 mg/kg to 650 mg/kg for fluoranthene, 55 mg/kg to 380 mg/kg for fluorene, 37 mg/kg to 300 mg/kg for phenanthrene, and 49 mg/kg to 1,300 mg/kg for pyrene. A quinoline representative, acridine, produced no toxicity at 1 mg/kg to 1,000 mg/kg. EC_{20} values demonstrated a large difference in sensitivity between plant species, with red clover being the most sensitive (Sverdrup, et al., 2003).

Fish and Invertebrates

With elevated sediment PAH levels, benthic organisms obtain a majority of their PAHs from sediments through their ability to mobilize PAHs from the sediment/pore water matrix. The elevated levels in the tissues of these benthic organisms could provide a significant source of PAHs to predatory fish. However, fish have the ability to efficiently metabolize and degrade PAHs. Food chain uptake of anthracene was studied using fathead minnows (*Pimephales promelas*) consuming water fleas (*Daphnia pulex*). The uptake was estimated at 15% of the amount accumulated from the water (Southworth 1979).

Toxicity of PAHs on earthworms is reported in Achazi and van Gestel (2003). The report LC_{50} concentration of >1,000 mg/kg anthracene in soil (2-week), >1,000 mg/kg chrysene in soil (2-week), >2,400 mg/kg fluoranthene in soil (3-week), 416 mg/kg fluoranthene in soil (4-week), 1,800 mg/kg fluorine in soil (3-week), 197 and 173 mg/kg fluorene in soil (2-week), and 69 mg/kg fluorene in soil (4-week), >2,000 mg/kg phenanthrene in soil (3-week), 134 mg/kg phenanthrene in soil (4-week), >2,300 mg/kg pyrene in soil (3-week) and 155 mg/kg pyrene in soil (4-week). *Eisenia veneta* and *E. eugeniae* are more sensitive to PAHs than *E. crypticus* and *E. fetida*.

Terrestrial Vertebrates

Mammals

PAHs can be readily absorbed through the inhalation, oral, and dermal pathways. Eisler (1987) reported LC_{50} values for rodents (*Rattus* spp. and *Mus* spp.) as 50 mg/kg-day benzo(a)pyrene, 700 mg/kg-day phenanthrene, and 2,000 mg/kg-day fluoranthene. Sublethal effects manifested as decreased pup weight in mice are reported at 10 mg/kg-day benzo(a)pyrene (MacKenzie and Angevine, 1981). Subchronic and chronic effects of exposure to PAHs in rats include liver and kidney damage, unspecified changes in peripheral blood pattern, body weight loss, genetic aberrations, and increased serum aminotransferase activity (Knobloch et al. 1969). Oral exposure to 120 mg/kg-bw/day benzo(a)pyrene resulted in a decreased survival time in two strains of mice (Robinson et al. 1975). Two oral studies in mice and one in rats indicated that benzo(a)pyrene induces reproductive toxicity in animals (Mackenzie and Angevine 1981; Rigdon and Neal 1965).

Birds

In a dietary toxicity tests, Patton and Dieter (1980) fed mallard 4,000 mg PAHs/kg diet (mostly naphthalene, naphthenes, and phenanthrenes) for seven months without adverse effects. Trust et al. (1994) report a 5-day LOAEL of 20 mg/kg body weight per day and NOAEL of 2 mg/kg body weight per day 7,12-dimethylbenz(a)anthracene oral toxicity in

European starling. Acute toxicity, LD_{50}, of acenaphthene, fluorine, anthracene, and phenanthracene in red-winged blackbird is 101, 101, 111, and 113 mg/kg body weight. The acute LD_{50} for anthracene in house sparrow is 244 mg/kg body weight (Schafer et al. 1983).

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SILVER

Environmental Fate and Transport

Sources

Silver is a naturally occurring soft metal used to make jewelry, silverware, electronic equipment, and dental fillings. It is mined concurrently with copper, lead, zinc, and gold (Grayson 1978). Because it is a rare metal, much of it is recycled. Silver may occur in nature in association with other elements such as nitrate (silver nitrate), oxygen (silver oxide), sulfur (silver sulfide), and chlorine (silver chloride). Silver compounds are used in photographic materials, which account for the bulk of silver emissions to the environment. Natural releases of silver occur through erosion (Scow et al. 1981).

Silver, in its pure form, is a lustrous, white solid that is insoluble in water, but soluble in nitric acid. Its melting point is 961^{o}C and molecular weight is 107.868. Silver compounds are white or charcoal colored and are either crystalline or powder. They melt at lower temperatures than pure silver, usually in the range of 200-400^{o}C (ATSDR 1990).

Transport and Fate

Silver released to the atmosphere occurs as an aerosol, and consists primarily of metallic silver, silver sulfide, silver carbonate, and silver halides (Smith and Carson 1977). Small particles in the aerosol may be transported by air currents, whereas larger particles may be deposited on the earth's surface through wet deposition or gravitational settling (Scow et al. 1981; Davidson et al. 1985).

The major forms of silver in water are silver sulfate, silver bicarbonate, and silver sulfate salts which are complexed with other ions (Boyle 1968). Most of the silver compounds which reach the water adsorb to particles and are deposited in aquatic sediments (Callahan et al. 1979).

Silver tends to complex with particles and organic matter in soils (Smith and Carson 1977); however, variables such as pH, clay concentration, particle size, and drainage affect the mobility of soil-bound silver and silver compounds.

Aquatic Wildlife

Silver tends to bioconcentrate in limited amounts in algae, mussels, and other aquatic organisms. Silver is toxic to soil microorganisms (Domsch 1984); however, it apparently bioaccumulates in marine algae (Fisher et al. 1984). Studies of bottom-dwelling species such as clams, oysters, and scallops indicate that these species also bioaccumulate silver (Thomson et al. 1984; Pesch et al. 1977). Bioconcentration within the food chain has not been demonstrated, but may potentially occur.

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Terrestrial Wildlife

Exposure to silver and silver compounds can occur orally, dermally, or by inhalation. Silver is found in most tissues, but has no known physiologic function. In long-term oral studies with experimental animals, silver compounds have produced slight thickening of the basement membranes of the renal glomeruli, growth depression, shortened lifespan, and granular silver-containing deposits in skin, eyes, and internal organs (Matuk et al. 1981; Olcott 1948, 1950). Hypoactivity was seen in rats subchronically exposed to silver nitrate in drinking water (Rungby and Danscher 1984). Mice that were orally administered doses of silver showed a decrease in weight gain (ATSDR, 1990).

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ZINC

Environmental Fate and Transport

Sources

Zinc is found in nature as ores of sulfide (sphalerite), oxide (franklinite), and carbonate (smithsonite). It is never found free and therefore metallic zinc must be obtained by extraction from ore. References to the use of zinc ores date back to biblical times (BC); however it was not recognized as a distinct element for ages. Zinc is used in the production of brass, alloys, die castings, and electrical conductors. Zinc oxide is used in the production of a variety of common items, including paint, rubber products, cosmetics, pharmaceuticals, soap, textiles, etc. (Weast, 1985; Sittig, 1980).

Zinc is a bluish-white metal, which is brittle at ambient temperatures, but malleable at 100-150^{o}C. It has a molecular weight of 65.38, a melting point of 419.58^{o}C, and a boiling point of 907^{o}C. Its specific gravity is 7.133 (Weast, 1985).

Fate and Transport

The concentration of zinc in soil porewater depends on soil pH, zinc forms, contents of clays, and minerals, organic matter, and other factors. Zinc becomes more soluble with decreasing soil pH and hence more mobile and bioavailable in acidic soil conditions, particularly at pH < 5 (Duquette and Henershot, 1990). In soils with pH > 7.7, Zn (OH)2 becomes the dominant form and solubility is very low. Zinc is a soluble form, such as zinc sulfate, is fairly mobile in most soils. However, relatively little land-disposed zinc is in soluble form, and mobility is, therefore, limited by a slow rate of dissolution. Consequently, movement towards groundwater is expected to be accompanied by corrosive substances (such as in mine tailings) (United States Environmental Protection Agency [EPA], 1980). Yet, soil conditions not suitable for zinc sorption may lead to leaching. Low pH (<7) and high ionic strength of the leaching solution favor desorption (EPA, 1987; Saeed and Fox, 1977).

Speciation and Bioavailability

Zinc is an essential element to both plants and animals. The active zinc species in the adsorbed state is the singly charged zinc hydroxide species (i.e. $Zn(OH)^{+}$ (Sanders and El Kherbawy, 1987). For calcareous soils, the relationship between zinc solubility and pH is nonlinear. At a high pH, zinc in solution is precipitated at $Zn(OH)_2$, zinc carbonate ($ZnCO_3$), or calcium zincate (Saeed and Fox. 1977). Clay and metal oxides are capable of sorbing zinc and tend to retard its mobility in soil.

The amount of bioavailable zinc will be determined by the amount of zinc present, which is soluble or may be solubilized. Plant uptake, losses by leaching, input of zinc in various forms, changes in moisture content in soil, pH changes, mineralization or organic matter and changing redox potential of the soil will influence the equilibrium. Due to the complexity of zinc interactions in soil, zinc transport behavior in soil cannot be predicted accurately (Hinz and Selim, 1994).

Zinc availability decreases as pH increases (Christensen, et al., 1992; Rehm and Schmitt, 1997); usually, increased zinc levels occur in soils with pH < 5.0 (Vitosh et al., 1994). Killorn (1984) reports that highly organic soils both increase and decrease zinc availability to plants. In addition, zinc availability decreases in cool soil temperatures (Killorn, 1984;) Rehm and Schmitt, 1997; Mahler et al., 1981). Furthermore, copper, iron, and manganese can inhibit plant uptake of zinc (Heckman, undated). Plant species have different tolerances levels to the availability of zinc. Grasses can tolerate high levels of available zinc while vegetables are sensitive (Vitosh et al., 1994). For example, fruit trees and corn are very sensitive to zinc deficiency, but carrots and peas have low sensitivity to zinc deficiency (Heckman, undated).

As with most metals, processing may cause aerosols to be released into the air as dust. Burning of coal tar may also release zinc to the atmosphere. These particles eventually settle out by gravitational forces or through deposition by precipitation. Zinc may be slowly oxidized in air; a process which is accelerated by moisture (Bowen, 1979; Sittig, 1980).

Zinc may form organic complexes in fresh water and settle in aquatic sediments; however sedimentation is slower than for some metals. In polluted estuaries zinc becomes desorbed from suspended matter, whereas in pristine estuaries zinc may remain sorbed to particles and become trapped in sediments (Bowen, 1979).

Dietary zinc absorption is highly variable in animals; in general, it increases with low body weight and low zinc status and decreases with excess calcium or phytate and by deficiency of pyridoxine or tryptophan. Low molecular weight proteins called metallothioneins play an important role in zinc homeostasis and in protection against zinc poisoning; zinc is a potent inducer of metallothioneins. Zinc interacts with many chemicals to produce altered patterns of accumulation, metabolism, and toxicity; some interactions are beneficial to the organism and others are not, depending on the organism, its nutritional status, and other variables. (Eisler, 1993).

Toxicological Profile

Plants

Zinc is a micronutrient for plants and is required to sustain regulation of growth, chlorophyll synthesis, carbohydrate formation, regulate enzymatic reactions and hormonal functions. At higher concentration, however, zinc could produce toxic effects in exposed organisms. The toxicity of zinc in ecosystems has been well documented in the available literature. The studies readily available on zinc plant toxicity cover a variety of endpoints. Small amounts (3.3 mg/kg) have been shown to decrease the annual ring growth of trees (Hagemeyer et al.,1993). At relatively low levels of 25 and 50 mg/kg, zinc has the effect of decreasing seed yields (Sheppard et. al., 1993) Aery and Sakar (1991). At higher levels, decreased leaf and plant weights and repressed grain yields are observed. Most plant studies use zinc sulfate.

Zinc has its primary effect on zinc-dependent enzymes that regulate RNA and DNA. In many types of aquatic plants and animals, growth, survival, and reproduction can all be adversely affected by elevated zinc levels. The most sensitive aquatic species were adversely affected at nominal water concentrations between 10 and 25 µg/L, including representative species of plants (Eisler, 1993). For freshwater plants, Suter and Tsao (1996) reported the lowest chronic screening value as 30 µg/L.

Invertebrates

Invertebrate studies are available for earthworms, along with assortment of other organisms. Almost all of the earthworm studies resulted in a decrease in cocoon production of growth rate at levels spanning from 136 mg/kg to 300 mg/kg. Effects on other invertebrates included death, decreased population size and decreased growth.

In many types of aquatic plants and animals, growth, survival, and reproduction can all be adversely affected by elevated zinc levels. The most sensitive aquatic species were adversely affected at nominal water concentrations between 10 and 25 µg/L, including representative species of protozoans, sponges, mollusks, crustaceans, and echinoderms (Eisler, 1993). For freshwater daphnids and non-daphnid invertebrates, Suter and Tsao (1996) reported the lowest chronic screening values as 46.73 µg/L and >5,243 µg/L, respectively.

Fish

In many types of aquatic plants and animals, growth, survival, and reproduction can all be adversely affected by elevated zinc levels. Zinc has its primary effect on zinc-dependent enzymes that regulate RNA and DNA. The gill epithelium is a primary target site in fish. The most sensitive aquatic species were adversely affected at nominal water concentrations between 10 and 25 µg/L, including representative species of fish and amphibians (Eisler, 1993). Suter and Tsao (1996) reported the lowest chronic screening value for freshwater fish to be 36.41 µg/L.

Terrestrial Vertebrates

Zinc has its primary effect on zinc-dependent enzymes that regulate RNA and DNA. The pancreas and bone are primary targets in birds and mammals.

Mammals

Mammals, compared to birds, are relatively resistant to zinc, as judged by their tolerance of extended periods on diet containing greater than 100 times the minimum daily zinc requirement. But excessive zinc exposure through inhalation or ingestion harms mammalian survival, metabolism and well being. The most sensitive species of mammals were adversely affected at dietary concentrations of 90 to 300 mg Zn/kg BW, drinking water concentrations greater than 300 mg Zn/kg BW, and air concentrations greater than 0.8 mg Zn/m^3 (Eisler, 1993).

Birds

Elevated zinc levels can cause mortality, pancreatic degradation, reduced growth, and decreased weight gain in birds. Pancreatic degeneration occurred in ducks fed diets

containing 2,500 mg Zn/kg ration. Ducks died when fed diets containing 3,000 mg Zn/kg feed or when given oral doses greater than 742 mg Zn/kg BW. Domestic poultry are routinely fed extremely high dietary levels of 20 g Zn/kg ration as a commercial management technique to force the molting of laying hens and the subsequent improvement of long-term egg production that molting produces. However, poultry chicks died at 8 g Zn/kg diet, had reduced growth at 2-3 g Zn/kg diet, and experienced pancreas histopathology when fed selenium-deficient but zinc adequate (100 mg Zn/kg) diets (Eisler, 1993).

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